This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
P446 .F34 1897 STOR
The disorders of digestion in infancy an
24503360365

LANE

MEDICAL LIBRARY

SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY

1885

Gift of

Dr. J. J. Tully.

DISORDERS OF DIGESTION

BY THE SAME AUTHOR.

The Dyspepsia of Phthisis. Its Varieties and Treatment. 8vo. 6s.

LONDON: H. K. LEWIS.

WITH DR. SAMUEL FENWICK.

Outlines of Medical Treatment. Fourth edition. 10s. 6d. 1894.

LONDON: J. & A. CHURCHILL.

THE

DISORDERS OF DIGESTION
IN
INFANCY AND CHILDHOOD

Property of Dr. J. J. Tully,
Stockton, Cal.

BY

W. SOLTAU FENWICK, M.D., B.S. LOND.

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS; PHYSICIAN TO OUT-PATIENTS AT THE EVELINA HOSPITAL FOR SICK CHILDREN

WITH ILLUSTRATIONS

LONDON: H. K. LEWIS
PHILADELPHIA: J. B. LIPPINCOTT COMPY.
1897

P446
F34
1897

[illegible]

TO THE

PRESIDENT

AND

MEMBERS OF THE COUNCIL

OF THE

BRITISH MEDICAL ASSOCIATION,

IN ACKNOWLEDGMENT OF THE ASSISTANCE RENDERED TO HIM

BY THEIR GRANTS FOR SCIENTIFIC RESEARCH,

THIS SMALL VOLUME

IS RESPECTFULLY DEDICATED

BY THEIR LATE RESEARCH SCHOLAR,

THE AUTHOR.

00255

PREFACE.

FOR several years the author has been much interested in the subject of dyspepsia in children, and it is mainly from the notes of some five thousand cases of disordered digestion which have come under his notice at the Evelina Hospital and elsewhere that the present volume has been compiled. The first five chapters deal with the physiology of digestion in early life, and the various diseases which are apt to arise from neglect of those fundamental laws which should regulate the diet and hygiene of infancy. The rest of the book is occupied with a description of the diseases of the stomach which are encountered during the period of childhood. The appendix includes a short account of the methods employed in the chemical examination of the contents of the stomach, and certain recipes and formulæ, to which special reference is made in the text. Most of the pathological work was done at the laboratories of the Royal College of Physicians and

Surgeons during the time that the author held the appointment of Research Scholar to the British Medical Association. The author would offer his grateful thanks to his father, Dr. Samuel Fenwick, for the loan of the drawings which are shown in Figs. 11, 12, 13, 14, and 15, and also to his friend, Mr. Noel Clarke, for his kind supervision of the work during its passage through the press.

W. SOLTAU FENWICK.

8, DEVONSHIRE STREET,
PORTLAND PLACE,
March 1, 1897.

CONTENTS.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE STOMACH IN INFANCY.

PAGE

Development and anatomical relations—Capacity—Methods of determination—Variations at different ages—Histology—Solitary glands—The gastric secretion—Variations in the amount of hydrochloric acid—Physiology of gastric digestion—Motor activity—The saliva and its functions—The pancreatic secretion—The bile—Micro-organisms of the stomach and intestines—The gases of the stomach and products of fermentation - - - - - 1—17

CHAPTER II.

DIET IN INFANCY.

Natural feeding—Secretion of milk—Variations in quantity—Variations in quality—Rules of nursing—Refusal of the breast—Contra-indications to nursing—Mixed feeding—Weaning—Diet in lactation—The wet-nurse—Artificial feeding—Variations in composition of milk—Differences between cow's milk and human milk—Care of the milk—Modification of milk—Simple dilution—Artificial human milk—Cream mixtures—Diet sheets—Preserved milks—Dried milks—Size and frequency of the meals—Sterilization of the food—Pasteurization—Sterilization—Feeding apparatus—Intolerance of milk—Strippings—Whey—Peptonized foods—Malted foods—Diet after sixth month—Diet after twelfth month—Diet after eighteenth month—Diet sheets - - - - - - - 18—58

CHAPTER III.

THE DYSPEPTIC CONDITIONS OF INFANCY

PAGE

Etiology — Symptoms — Pain and flatulence — Nausea and vomiting — Diarrhœa — Green stools — Fat diarrhœa — Appetite—Tongue—Urine—Temperature—Loss of flesh — Pulse — Reflex symptoms — Chemistry of digestion — Progress and termination — Complications — Diagnosis — Treatment - - - - - - 59—90

CHAPTER IV.

ACUTE GASTRO-INTESTINAL CATARRH IN INFANTS.

Etiology — Simple and infective forms — Epidemic variety — Mode of infection—Morbid anatomy—Morbid histology —Bacteriology—Symptoms—Mode of onset—Vomiting— Pain—Diarrhœa — Progress and termination—Complications — Diagnosis — Prognosis — Treatment — General — Dietetic—Medicinal—Gastro-intestinal antisepsis - 91—149

CHAPTER V.

CHRONIC GASTRO-INTESTINAL CATARRH IN INFANTS.

Etiology—Morbid anatomy—Morbid histology—The stomach — Intestine — Brain — Kidney — Liver—Symptoms—Complications and sequelæ—Physical examination—Chemistry of digestion — Examination of the blood—Progress and termination — Prognosis—Diagnosis—Treatment—General —Dietetic—Medicinal - - - - 150—212

CHAPTER VI.

ACUTE GASTRIC CATARRH IN CHILDREN.

Etiology — Hereditary and acquired predisposition — Acute primary catarrh—Secondary catarrh—Membranous gastritis —Morbid anatomy—Morbid histology—Acute interstitial gastritis—Parenchymatous gastritis—Casts of the gastric glands—Clinical varieties—Acute indigestion or embarras gastrique—Biliousness—Recurrent gastric catarrh—Cyclical vomiting—Toxic gastritis—Febrile gastric catarrh—The

PAGE

epidemic variety—Physical examination—Chemistry of digestion—Diagnosis—Treatment—General—Dietetic—Medicinal - - - - - - - 213—247

CHAPTER VII.

CHRONIC GASTRIC CATARRH IN CHILDREN.

Etiology—Morbid anatomy—Morbid histology—Chronic parenchymatous gastritis—Chronic interstitial gastritis—Symptoms—Chemistry of digestion—Diagnosis—Treatment—General—Dietetic—Medicinal - - - 248—261

CHAPTER VIII.

WEAK DIGESTION IN CHILDREN.

Etiology — Predisposition — Symptoms — Complications — Chemistry of digestion—Treatment—General—Dietetic—Medicinal - - - - - - - 262—279

CHAPTER IX.

ULCERATION OF THE STOMACH.

Frequency — Varieties — Hæmorrhagic erosions — Follicular ulceration—Its pathology and causes—Simple ulceration—Acute perforating ulcer—Cases—Simple chronic ulcer—Cases—Etiology of simple ulcer—Causes of non-healing—Tubercular ulcer—Cancer of the stomach—Symptoms of gastric ulcer—Pain—Vomiting—Hæmatemesis—Melæna—Melæna neonatorum—Other symptoms—Complications—Perforation—Dilatation of the stomach—Congenital stenosis of the pylorus—Its pathology and symptoms—Treatment of gastric ulcer - - - - 280—318

CHAPTER X.

THE DYSPEPSIA OF STRUMOUS CHILDREN.

Sex and age—Family history—General appearance—Tendency to tuberculosis—Symptoms—Pain—The bowels—The appetite—Thirst—Gastric catarrh—Relapses—Etiology—Treatment - - - - - - - 319—333

CHAPTER XI.

PAROXYSMAL HYPERACIDITY.

PAGE

Causes—Sketch of a case—Relative frequency—Symptoms—Character of the gastric contents—Variations in severity—Gastroxynsis—Gastroxie—Vomitus hyperacidus—Etiology—Treatment - - - - - - 334—342

CHAPTER XII.

DYSPEPSIA DEPENDENT UPON DISEASE OF OTHER ORGANS.

Pulmonary disease—Tuberculosis—Chronic phthisis—Initial symptoms—Morning sickness—Reflex cough—Appetite—Terminal dyspepsia—Pathology—Treatment. Cardiac disease—Pathology—Dyspeptic symptoms—Physiology—Treatment. Renal disease—Gastric catarrh—Varieties—Etiology—Symptoms—Treatment. Nervous disease—Pathology—Symptoms—Treatment - - - 343—359

APPENDIX.

Chemical examination of the contents of the stomach—Test-meals—Total acidity—Detection of hydrochloric and organic acids—Quantitative analysis—Free and combined acids—Pepsin—Rennet—Selected recipes—Prescriptions - - - - - - - 361—369

INDEX - - - - - - - 371—377

LIST OF ILLUSTRATIONS.

FIG. PAGE
1. Section of a Normal Infant's Stomach - - 5
2. Section of the Stomach, showing an Enlarged Solitary Gland in the Mucous Membrane - - - 6
3. Section of the Stomach of a Young Guinea-pig, showing a Solitary Gland situated beneath the Mucous Membrane - - - - - - 7
4. Section of the Stomach of a Healthy Child - 160
5. Section of the Stomach in an Early Case of Acute Gastro-intestinal Catarrh - - - - 161
6. Photo-micrograph of the Stomach in the Second Stage of Chronic Gastro-intestinal Catarrh - 163
7. Photo-micrograph of the Stomach in an Advanced Case of Chronic Gastro-intestinal Catarrh, showing Complete Cirrhosis of the Mucous Membrane - 165
8. Photo-micrograph of the Small Intestine in a Case of Chronic Gastro-intestinal Catarrh - - 167
9. Photo-micrograph of the Large Intestine in a Case of Chronic Gastro-intestinal Catarrh - - 169
10. Section of the Stomach in a Case of Acute Interstitial Gastritis - - - - - 221
11. Section of the Stomach in a Case of Scarlatina - 223
12. Casts of the Gastric Glands - - - - 224
13. Section of the Stomach in a Case of Measles - 225
14. Drawing of the Gastric Glands in the Second Stage of Chronic Parenchymatous Gastritis - - 251

FIG. PAGE
15. DRAWING OF THE MUCOUS MEMBRANE OF THE STOMACH IN AN ADVANCED CASE OF CHRONIC PARENCHYMATOUS GASTRITIS - - - - - - - 252
16. SECTION OF THE STOMACH IN A CASE OF CHRONIC INTERSTITIAL GASTRITIS - - - - - - 253
17. DRAWING OF THE PYLORIC END OF THE STOMACH, showing Inflammation of the Solitary Glands, with Acute Ulceration originating in these Structures (after Carswell) - - - - - - - - 284
18. SECTION OF A STOMACH AFFECTED WITH CATARRHAL INFLAMMATION, showing an Enlarged Solitary Gland - 285
19. SECTION OF THE STOMACH OF A GUINEA-PIG, showing an Enlarged Solitary Gland with Inversion of the Mucous Membrane - - - - - - - 285
20. SECTION OF A STOMACH AFFECTED WITH FOLLICULAR ULCERATION - - - - - - - 286
21. DRAWING OF A STOMACH IN A CASE OF ENTERIC FEVER, showing Four Perforating Ulcers in the Neighbourhood of the Pylorus - - - - - - - 287

DISORDERS OF DIGESTION
IN INFANCY AND CHILDHOOD.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE STOMACH IN INFANCY.

In the fœtus the first sign of the differentiation of the stomach from the primitive alimentary canal consists of an abrupt deviation of the tubular gut toward the right side of the body, the concave or upper border of the loop thus formed constituting the lesser, and the lower border the greater, curvature of the future organ. Finally, the loop is turned over so as to lie upon what was previously its right side, which now becomes the posterior surface. Owing to this peculiar process of development, the stomach at birth presents several important differences from the adult organ which are well worthy of notice.

ANATOMY.

The absence of the pouch-like fundus which characterizes the stomach in later life causes the infantile organ to present a globular or slightly oval form, the long axis of which passes obliquely downwards and to the right across the median line of the body. The œsophagus enters the stomach in an abrupt manner, its lower end being somewhat expanded and the mucous membrane thrown into numerous longitudinal folds, which can be traced for some distance into the viscus. The cardiac orifice is situated on the left side of the spine, opposite the body of the tenth dorsal vertebra, to which it is usually adherent. From this point the lesser curvature descends perpendicularly along the left border of the spine for the first half of its extent, and then turns to the right, so that its lower portion lies transversely across the bodies of the vertebræ. The pylorus, which is situated on a lower plane than the cardia, lies completely under cover of the left lobe of the liver, and, when the organ is empty, usually occupies a spot midway between the tip of the ensiform cartilage and the navel, and slightly to the left of the median line. Owing to the comparatively fixed position of the cardia, and the deficient development of the lesser omentum, the pyloric end of the stomach is capable of considerable movement, so that, when the stomach becomes filled with food, it is dragged downwards and to the left, describing in its progress a kind of ellipse, the centre of which coincides with the œsophageal opening in the diaphragm. In its distended condition the stomach can be mapped out upon the surface of the body with considerable exactitude. The cardiac orifice corresponds to the sixth left costal cartilage at the point where it is intersected by the left mammary line. The lesser curvature is represented by a line drawn along the upper border of the sixth rib as far as the left anterior axillary line, and thence across the axilla along the lower border of the seventh rib. The inferior border of the organ corresponds to a line drawn transversely across the left side of the abdomen from a spot midway between the tip of the ensiform cartilage and the umbilicus (Henschel).[1]

CAPACITY.

The capacity of the stomach at different periods of infant life is a matter of considerable importance from a dietetic point of view, and has been made the subject of numerous investigations (Frolowsky,[2] Fleishmann,[3] Holt,[4] Rotch[5]). One method of determining the gastric capacity of a child is to observe the increase of the body-weight which follows the ingestion of a full meal. For this purpose the infant is carefully weighed in the fasting state, and then permitted to take the breast until its appetite is satisfied. It is then weighed again, and the initial result deducted from the final one. Since one fluid ounce of human milk weighs about one ounce, the increase in the body-weight represents fairly accurately the volumetric capacity of the stomach. Another method, which has been extensively employed by Holt and others, consists in ligaturing the two orifices of the stomach after death without disturbing the anatomical connections of the organ, and then injecting a quantity of water just sufficient to obliterate the rugæ of the mucous membrane. From these various experiments the following conclusions may be drawn : At birth the average capacity of the stomach varies from fivesixths to one fluid ounce. During the first three months of life the organ undergoes a rapid increase in size, the average monthly increment being about one fluid ounce. From the third to the eighth month the growth of the organ is much slower, so that the average monthly increase in its capacity is only about half an ounce; while between the eighth and the fourteenth months the monthly gain is about one-third of an ounce. The absolute capacity of the organ at any age also varies directly with the weight of the body, and, as Fleishmann has shown, is somewhat greater in hand-fed than in breast-fed infants. These results are expressed more fully in the following table (Holt):

---

[1] *Archiv. f. Kinderheilk.*, xiii., p. 32, 1891.

[2] Inaug. Dissert., St. Petersburg, 1876.

[3] *Klinik der Pädiatrik*, 1875.

[4] *Archiv. of Pediatrics*, vii., p 960, 1890.

[5] 'Lectures on Pediatrics,' vol. i., p. 79, 1896.

TABLE I.

Age of Infant.	Capacity of Stomach (fluid ounces).
Birth	$\frac{5}{8}$-1
2 weeks	1·5
1 month	2·5
2 months	3·37
3 "	4·5
4 "	5·6
6 "	5·9
8 "	7·66
10 "	8·4
14 "	8·9

HISTOLOGY.

In its histological structure the stomach of an infant does not differ materially from that of an adult. The gastric glands are well formed, and both the central and parietal cells can easily be discerned. It is usually noticeable, however, that the mouths of the glands are somewhat dilated, while the ducts present a greater diameter than is usual at a later period of life (Fig. 1).

The mucus-secreting cells are also increased in number, and occasionally the columnar epithelium, which normally covers the inner surface of the organ, is replaced by goblet cells. When examining the stomachs of kittens and other young animals, I have often been struck by a similar appearance of functional activity in the superficial epithelium, and, by killing the animals at various intervals after a meal, have invariably found that the first effect of the introduction of food into the stomach is to cause a transformation of the columnar epithelium into cells of the goblet type. It would therefore appear that in early life the superficial epithelium of the stomach produces an abundant secretion of mucus, which serves to protect the delicate secretory structures from irritation by the food during the process of digestion. Another point

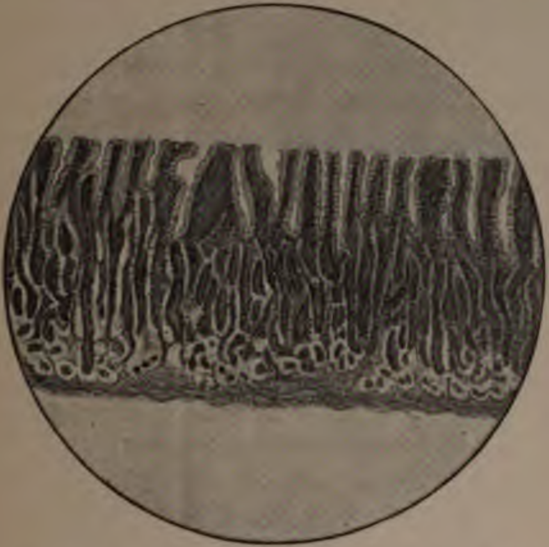

FIG. 1.—Section of a Normal Infant's Stomach. (× 80.)

of interest in the stomachs of new-born children resides in the distribution of the lymphatic tissue. With the aid of the microscope two forms of lymphoid structures can be detected. In the simplest variety, the adenoid tissue merely consists of a few nucleated cells scattered here and there around the blind extremities of the gastric tubules, and occasionally spreading for some distance over the adjoining surface of the muscularis mucosæ. In the second and rarer variety the lymphatic tissue exists as circumscribed masses in the substance of the mucous membrane. These so-called 'solitary glands' are somewhat oval in shape, and rest upon the muscularis mucosæ like eggshells upon their broken ends (Fig. 2).

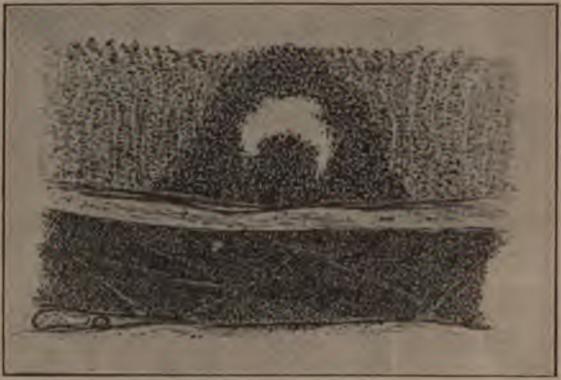

FIG. 2.—Section of the Stomach, showing an Enlarged Solitary Gland in the Mucous Membrane. (× 80.) (The centre has fallen out during the process of preparation.)

At the circumference of the mass the gastric tubules are displaced, and assume a slanting direction; but toward the free surface they regain their perpendicular position, and completely cover the superficial aspect of the gland. No limiting membrane can be discovered, but each gland is provided with a small nutrient artery, while from its deeper surface there passes a lymphatic vessel to join the larger trunks situated in the submucosa. Occasionally the base of the gland is situated in the submucous tissue, and extends thence into the mucous membrane through an aperture in the muscularis mucosæ. This hourglass form is common in some animals like the guinea-pig and rat, but is rarely encountered in the human infant, except in the neighbourhood of the lesser curvature (Fig. 3).

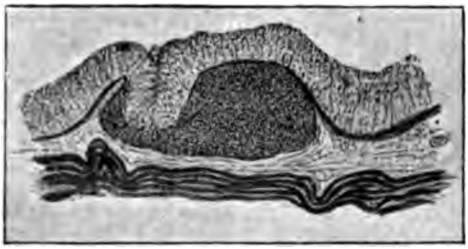

FIG. 3.—Section of the Stomach of a Young Guinea-pig, showing a Solitary Gland situated beneath the Mucous Membrane. (× 60.)

In the pig and other animals the solitary glands of the stomach can easily be discerned with the naked eye when the mucous membrane is dissected off the subjacent muscular coat, but in the human subject it is necessary to employ some special means of rendering them conspicuous. This is most readily effected by immersing the tissue for some hours in a dilute solution of glacial acetic acid, at the end of which time the solitary glands may be recognised in the form of small opaque specks in the midst of the swollen and gelatinous tissue; and if the surface of the mucous membrane has been previously mapped out into areas of a centimetre square, it is easy to determine the relative number of glands in the different regions of the organ. By the adoption of this method I have been enabled to arrive at the following conclusions concerning the size and relative number of the solitary glands at different periods of life. Up to the age of six months the glands are usually small and ill defined, except in the region of the lesser curvature. After this age they undergo gradual development, until about the age of ten years as many as five or six may be counted in every square centimetre of the mucous membrane in the fundus, and more than twice this number in the vicinity of the pylorus. From puberty onwards the solitary glands begin to disappear from the central regions of the stomach, so that after the age of forty it is often impossible to demonstrate their presence in the cardiac two-thirds of the viscus without the aid of a microscope, although a peculiar pitted appearance of the surface often betokens the former existence of a lymphoid follicle.

It is also worthy of notice that the muscular tunic of the stomach does not attain its full degree of development until about the tenth month of extra-uterine life. Before this age both the longitudinal and circular layers of fibres are remarkably thin, while the oblique fibres are often wanting altogether. The circular fibres which constitute the sphincter of the cardiac orifice are also very deficient.

PHYSIOLOGY.

The Gastric Secretion.—Although the stomach at birth possesses secretory powers sufficient to ensure the adequate digestion of the milk it is destined to receive, it does not exhibit the same degree of activity as in later life. In order, therefore, to appreciate the changes which take place in the gastric secretion as the result of diseased conditions of the stomach, it is necessary to comprehend the manner in which digestion is carried on in the stomach of a healthy infant, and the variations to which it is liable under normal circumstances.

It is allowed by all that hydrochloric acid is a necessary component of the gastric juice, and that it appears in the contents of the stomach coincidently with the first meal. Concerning. however, the quantity of the acid, and the presence of *free* acid during the course of normal digestion, many contradictory opinions have been advanced. Thus, while some writers assert that free hydrochloric acid is never found in the infant's stomach, others believe that its presence can occasionally be detected, while others, again, maintain that free acid at the termination of digestion is the rule and not the exception. In like manner, the occurrence of lactic acid in the gastric contents has been both attested and denied. With the hope of being able to throw some light on these and other disputed points, I instituted a series of experiments upon healthy infants, some of whom were fed entirely from the breast, while others received either cow's milk or some form of farinaceous food. In each instance the same quantity of food was administered, and the contents of the stomach were withdrawn after a definite interval of time, and submitted to the same chemical tests. From the results thus obtained, the following conclusions have been drawn :

1. The quantity of hydrochloric acid secreted varies in different children, and also in the same child from day to day and from meal to meal.

In healthy infants *at the breast* the milk is usually found to be curdled after a residence of ten to fifteen minutes in the stomach. This depends upon the presence of rennet in the gastric secretion, and may be observed immediately after birth.

The *acidity* of the gastric contents gradually increases during the progress of digestion, and attains its maximum from ninety to one hundred and ten minutes after the commencement of the meal. The average *total acidity* (calculated as hydrochloric acid) at the end of the first ten minutes is about 0·02 per cent. ; at the end of an hour, 0·06 to 0·075 per cent. ; and after eighty minutes, 0·13 per cent. It must be noted, however, that the ordinary methods of filtration lessen the acidity of the gastric contents very considerably, the difference between the filtered and crude material often amounting to as much as 0·05 per cent.

Free hydrochloric acid is an inconstant factor, and can seldom be detected until after the expiration of eighty minutes. It can usually be recognised, however, when the organ has become partially empty.

Pepsin is invariably present as long as the secretion contains any trace of the mineral acid.

Lactic and other secondary acids do not occur as a normal product of digestion, and their appearance in the contents of the stomach must consequently be regarded as evidence of fermentation.

2. In infants fed upon *cow's milk*, the total acidity of the gastric contents is more pronounced than in breast-fed infants, and often amounts at the end of eighty minutes to as much as 0·18 per cent. HCl.

Free hydrochloric acid can usually be detected toward the latter end of digestion. In the majority of cases lactic acid can also be detected, but it never exists in any appreciable amount.

3. In infants who are fed upon *farinaceous* foods, the total acidity of the gastric contents is invariably diminished, and may not exceed more than half the normal. In a few cases of this description, where I administered a trial meal, consisting of oatmeal and water, the contents of the stomach, extracted after eighty minutes, was only faintly acid to litmus-paper. The same children, when supplied with milk, exhibited the normal powers of secretion.

With these facts in view, it is possible to offer an explanation of some of the peculiarities of infantile digestion. The introduction of milk into the stomach at once excites the secretion of gastric juice, which is poured out in quantities that have direct relation to the size of the meal. Each successive atom of free acid which makes its appearance in the cavity of the stomach is immediately seized upon by the proteid elements of the food, and fixed by them in the form of a chemical combination. Pfungen[1] has shown by experiment that 100 grammes of milk are capable of thus 'fixing' or saturating 0·298 gramme of hydrochloric acid; and Lüttke[1] has demonstrated that this combination of proteid and acid not only remains stable when exposed to a high temperature, but also fails to exhibit the distinctive reactions of the free mineral acid with phloroglucin-vanillin and other colour reagents. These observations afford a rational explanation of the apparent absence of free acid from the stomach until the end of digestion, since it is obvious that it cannot be detected until all the proteids of the food have been saturated. It is well known that free hydrochloric acid exerts an inhibitive action upon the growth of micro-organisms when it exists in quantities greater than 0·17 per cent. (Miller); and during adult life the plentiful secretion of gastric juice probably performs a valuable antiseptic function. But in infancy, for reasons already explained, an excess of free acid is seldom observed, and consequently any bacteria which may gain an accidental entrance to the stomach with the food are enabled to flourish without restraint. This probably explains the extreme susceptibility of young infants to gastro-intestinal infection.

---

[1] *Wien. Klin. Wochen.*, s. 106, 1892.

The *motor activity* of the stomach can easily be investigated by observing the period of time required by the organ to digest and dispose of a full meal of milk. From experiments made in this direction, Epstein[2] came to the conclusion that in healthy infants at the breast the stomach is usually empty within an hour and a half; but Van Puteren,[3] Wohlman,[4] and Leo[5] appear to allow about two hours for the full performance of gastric digestion. In my own cases, the time was found to vary with the nature of the milk, being on the average one and a half hours in children at the breast, and two to two and a quarter hours in those who were fed on cow's milk. In all cases the major portion of the food disappears from the stomach during the first hour, about three-quarters of an hour usually being required for the adequate disposal of the last thirty or forty cubic centimetres of milk. Peptone can always be recognised in the contents of the organ within half an hour of the meal—a fact which disposes of the foolish statement that the stomach in infancy is not a digestive organ, but merely a reservoir for the food. After the organ has transmitted the whole of the meal into the small intestine, it does not remain absolutely empty, but usually contains a small quantity of mucus and gastric juice, in which free hydrochloric acid can be recognised. By means of certain experiments conducted with salol, Pfannenstill[5] has shown that the muscular movements of the stomach are not less active in infants than in older children, since salicyluric acid appears in the urine within the normal period of time.

---

[1] Martius and Lüttke, 'Die Magensäure des Menschen,' 1892.

[2] *Archiv. f. Kinderheilk.*, Bd. iv., s. 325, 1883.

[3] Inaug. Dissert., St. Petersburg, 1889.

[4] *Jahrb. f. Kinderheilk.*, xxxii., p. 297.

[5] *Berl. Klin. Wochensch.*, xlix., s. 981, 1888.

Nature having ordained that infants should subsist upon milk, it is not surprising to find that the various secretions, whose chief function is to transform starch into sugar, are invariably deficient in early life. Thus, the secretion of *saliva* is extremely scanty during the first two months, and only becomes abundant about the period of dentition. At birth the parotid gland alone contains the ptyalin ferment, and the mixed secretions of the mouth are found to exert but little action upon starch. About the eighth week the diastatic ferment can usually be detected in the secretion of the submaxillary gland; but it is not until the fourth month that the saliva exhibits any decided and constant influence upon starch. It is also worthy of notice that in inflammatory states of the buccal mucous membrane, especially the parasitic, the secretion of ptyalin is usually deficient (Zweifel).[1]

---

[5] *Jahrb. f. Kinderheilk.*, xxxi., p. 373.

The *pancreatic* secretion also lacks its diastatic ferment during the first two months of life, and it is usually not until about the end of the first year that the secretion of the gland exhibits its normal degree of activity (Korowin). Trypsin, on the other hand, appears to be constantly present in the pancreas at birth; and possibly the other principles in its secretion which curdle milk and split up neutral fats also exist in small quantities at an early period.

Bile appears to be manufactured by the liver as early as the third month of fœtal life, and accumulates in the intestine until expelled after birth in the meconium. The secretion continues active during the whole period of infancy, and imparts to the fæces their characteristic golden colour.

MICRO-ORGANISMS OF THE DIGESTIVE TRACT.

At birth the contents of the stomach and intestine are sterile; but within a few hours direct infection occurs through the medium of the air, which the infant swallows in large quantities (Breslau), and by the entrance of bacteria through the rectum (Escherich).

The Stomach.—Our knowledge of the bacteriology of the stomach in infancy has chiefly been derived from the observations of Van Puteren,[2] who carefully examined the gastric contents of forty healthy infants, ranging from four to seventy-seven days old, some of which were fed upon the breast, while others were nourished upon cow's milk. This observer found that the stomachs of children fed in an artificial manner usually contained about twenty times as many micro-organisms as infants at the breast, and that in cases of thrush the number of bacteria was always enormously increased. Among the various species which he was able to isolate, the *B. lactis aerogenes* occurred in 37·6 per cent. of the breast-fed, and 45 per cent. of bottle-fed infants; the *B. subtilis* in 11·7 per cent. of breast-fed, and 36·8 per cent. of bottle-fed; the *Oidium lactis* in 12·9 per cent. of breast-fed, and 27·3 per cent. of bottle-fed; the *Staphylococcus pyogenes aureus* in 16·4 per cent. of the breast-fed, and in 27·2 per cent. of the bottle-fed; non-liquefying cocci in 12·9 per cent. of the breast-fed, and 54·4 per cent. of the bottle-fed; while the *B. fluorescens liquefaciens* occurred in 24·3 per cent. of the bottle-fed, and the *B. butyricus* (Hueppe) in every case where the infant was nourished upon cow's milk. The relative number of these micro-organisms did not vary to any marked extent during the different stages of digestion, and the majority could be extracted from the stomach in an active state at the height of the process. Many of these bacteria could also be obtained from the organ in the intervals of digestion, so that it is obvious that the stomach in infancy, instead of being a 'sterilizing chamber,' must be regarded as a natural form of incubator, in which every species of micro-organism that finds an entrance with the food is afforded every facility for growth and multiplication.

---

[1] 'Untersuchungen ueber den Verdauungsapparat des Neugeborenen,' 1874.

[2] *Wratch.*, No. 22, 1888.

The Intestines.—The researches of Escherich[1] have shown that in an infant the intestinal bacteria vary with different kinds of food and in different regions of the bowel. The first to make their appearance are torulæ and cocci; but these are rapidly replaced by others as soon as the intestine becomes filled with milk. In the small intestine of breast-fed children, the most abundant and constant species is the *B. lactis aerogenes*, while in the large intestine the bacillus of Brieger occurs in enormous numbers. Among the other and rarer forms, the *B. subtilis*, *Proteus vulgaris*, and several varieties of cocci and streptococci, are occasionally observed. It is possible that the absence of oxygen is to a great extent responsible for the limited number of species; but it is also probable that the chemical products of the *B. lactis aerogenes* are not without an effect in inhibiting the growth of other and less active forms. With regard to the chemical action of these different micro-organisms upon the constituents of the milk, comparatively little is known. The *B. lactis aerogenes* converts the milk-sugar into lactic acid; but this property is also shared by other organisms which are habitually present in the stomach or intestine.

Butyric acid is manufactured from lactic acid by the action of the *B. butyricus*, free hydrogen and carbonic acid gas being liberated at the same time. It is also probable that alcohol and acetic acid are produced by the action of several bacteria, and that under certain conditions organic poisons may be manufactured in the digestive tract, which, when absorbed, are capable of exerting a specific action upon the nervous system.

---

[1] 'Die Darmbacterien des Säuglings,' s. 116, 1886.

The *gaseous* contents of the stomach consist of a mixture in varying proportions of hydrogen, nitrogen, and carbon dioxide gas, with occasional traces of marsh gas. The nitrogen is probably derived from the atmospheric air swallowed by the infant, while the remaining gases arise during the various processes of fermentation.

CHAPTER II.

DIET IN INFANCY.

ALMOST all the disorders of digestion which occur during the first two years of life are due to some fault either in the composition or mode of administration of the food. It may, therefore, not be out of place to preface a description of the morbid states of the alimentary canal by a brief account of those principles of infant feeding which constitute the only efficient remedy we possess against the incidence of disease.

In the mammary secretion of its mother Nature provides every new-born child with that form of nourishment which is best adapted to its digestive capabilities and functional requirements, and it is only when some accident renders this natural food deficient in quantity or injurious in quality, that it becomes necessary to have recourse to some other form of nourishment which in its chemical and physical composition is closely allied to human milk.

NATURAL FEEDING.

Secretion of the Milk.

The secretion of the breast for the first forty-eight hours after parturition presents an abnormally thick and yellow appearance, owing to the presence in it of a number of large corpuscles containing globules of fat. This early secretion, or colostrum, has a higher specific gravity than ordinary milk, and contains a relatively large amount of albumin and salts, but is deficient in fat and sugar. When imbibed by the infant, it acts as a laxative upon the intestine, and promotes the evacuation of the meconium. About the third day a change takes place in the character of the secretion, which now acquires a bluish-white colour, and presents a vast number of minute globules of fat; while the colostrum corpuscles rapidly diminish in number, and finally disappear from the milk about the second or third week of lactation. Milk when freshly drawn from the breast is slightly alkaline in reaction, and has a specific gravity of about 1031. It contains on an average 87 per cent. of water and 13 per cent. of solids. The greater part of the fat is composed of a mixture of palmitin, stearin, and olein, along with a small quantity (2 per cent.) of the glycerides of caproic, butyric, caprylic, and myristic acids. The presence of fat in the milk is essential to the nutrition of the infant, and probably helps also to maintain the temperature of the body. The proteids, or nitrogenous elements of the food, consist of caseinogen and lact-albumin, which vary in amount at different periods of lactation. The requisite amount of carbo-hydrate is supplied by the lactose, or 'sugar of milk,' which constitutes about half the total quantity of solids. The mineral ingredients consist of various salts of potash, soda, lime, magnesia, phosphorus, and alumina. In addition to these elements, milk also contains a substance which endows it with antiscorbutic properties of a high order.

Variations in Quantity.

The total quantity of milk secreted during the course of each day is apt to vary both under normal and abnormal conditions. As a rule, the breast of a healthy woman during the early stages of lactation produces from one and a half to two ounces of milk every two hours, the total yield for each breast being therefore eighteen to twenty-four ounces per diem. With the progress of lactation the amount is gradually increased, so that at the end of the ninth month the total quantity measures about sixty ounces each day. This increase in the mammary secretion bears a definite relation to the growth of the stomach and to the nutritive requirements of the infant; or, in a word, the supply is strictly adjusted to the demand. This fact is well shown when a comparison is made in a large number of normal cases between the supply of the breast milk and the weight of the infant; for it is found that at the same age the daily amount of milk produced varies directly with the body-weight, and consequently with the volumetric capacity of the infant's stomach.[1] After the ninth month the secretion of milk usually diminishes, and as soon as the child is permanently removed from the breast the secretion rapidly ceases. The daily quantity of milk may be *diminished*, owing to some abnormal condition either of the infant or its mother. Anything which interferes with the regular depletion of the breast, by depriving the organ of its normal stimulus to secretion, will necessarily lessen the supply of milk. Thus, failure of the secretion is frequently observed in cases where the infant, either from constitutional debility or from some morbid affection of the mouth, is unable to suck with sufficient strength and energy.

---

[1] See p. 4.

An impaired state of the mother's health adversely affects the secretion of her breast, and an attack of severe diarrhœa, influenza, or some acute febrile malady, is not infrequently followed by a suppression of the mammary functions. The quantity of the milk may also be diminished by nervous disturbances or by an absolute scarcity of food, while the administration of cathartic drugs, or such substances as belladonna and alum, to a nursing woman often gives rise to a marked diminution in the daily output of milk. An abnormal *increase* of the secretion (galactorrhœa) occurs under certain pathological conditions, but the influence of the so-called galactogogues in stimulating the flow of milk is extremely doubtful.

Variations in Quality.

From a practical point of view the variations which occur in the chemical composition of the milk are of more importance than those which concern the amount of the secretion, since it is upon them that most of the disorders of digestion depend.

Analysis of the milk of a nursing woman shows that it varies considerably in composition at different periods of lactation. As a rule, the amount of caseinogen steadily increases until the third month, after which time it remains fairly constant. The percentage of sugar does not vary materially after the first week or two, while the mineral constituents of the milk increase in quantity until the end of the fifth month, and exhibit a constant ratio to the percentage of proteids. The fat is the most inconstant of all, and varies in quantity from week to week even in the same woman under normal conditions (Pfeiffer).

Reiset and Peligot have shown that the milk varies considerably in composition during the process of milking, that portion which is first drawn being abnormally watery, while the last part of the secretion contains an excess of solid material, of which the principal ingredient is fat. The composition of the milk also varies according to the length of time which is allowed to elapse between successive milkings. Too frequent nursing lessens the amount of water and increases the percentage of solids, while infrequent evacuations of the breast causes the secretion to become too watery, and consequently less nutritious than normal. The exact composition of the milk depends to a great extent upon the nature of the food supplied to the woman and the amount of exercise in which she indulges. Thus, a diet containing an excess of proteid food, combined with a deficient amount of physical exercise, is productive of a milk containing an undue percentage of albuminous and fatty material; while a limited consumption of nitrogenous food and excessive exercise usually gives rise to a watery milk deficient in solids.

When the milk of several healthy women is submitted to analysis, it is found that the various specimens differ markedly among themselves in the percentage value of their principal constituents. It must therefore be concluded that the mammary secretion of each woman is especially adapted to the digestive capabilities of her own child, and that the general health of her offspring must be considered as the only reliable indication of the dietetic value of her milk. Diseased states of the mother, both functional and organic, are apt to alter the composition of the milk in such a manner as to render it less digestible or less nutritious than normal. Temporary alterations of this nature may be induced by severe mental emotions like anger, fright, or anxiety, or by an attack of some acute disorder, such as tonsillitis, diarrhœa, mumps, etc. In such cases the milk is usually rendered difficult of digestion by an increase in the amount of its proteids, although, according to Becquerel, the percentage of fat may likewise be augmented. The reappearance of the catamenia in a nursing woman is often accompanied by a temporary alteration of a similar nature, while pregnancy occurring during the course of lactation is apt to be followed by a more enduring alteration in the quality of the milk. In the latter case, however, the secretion more often exhibits a deficiency of solids than the opposite condition. These variations in the quality of the milk are well shown in the following table taken from Rotch's 'Lectures upon Diseases of Infants':

TABLE II.

	Normal Milk.	Poor Milk (starvation).	Over-rich Milk (rich feeding, lack of exercise).	Bad Milk (pregnancy, disease, etc.).
Fat	4·0	1·1	5·1	0·8
Sugar	7·0	4·0	7·5	5·0
Proteids	1·5	2·5	3·5	4·5
Ash	0·15	0·09	0·2	0·09
Total Solids	12·65	7·69	16·3	10·39
Water	87·35	92·31	83·70	89·61
	100·00	100·00	100·00	100·00

Finally, it must be mentioned that the breast, especially if its function is deranged, acts as an excretory as well as a secretory organ, and consequently, if the mother should indulge in such drugs as mercury, iodide of potassium, colchicum, morphine, lead, and arsenic, her milk is liable to be contaminated by them, and to produce toxic symptoms in the infant. Saline medicines and the compound powder of liquorice occasionally confer a purgative property upon the mammary secretion, while certain articles of diet, like oatmeal and beer, when taken by the mother, may induce dyspeptic symptoms in the suckling from the same cause.

All chronic diseases affecting the mother, such as phthisis, cancer, renal inflammation, etc., render the milk either innutritious or indigestible. To this rule, however, syphilis is usually an exception.

The Rules of Nursing.

The infant should be put to the breast a few hours after birth, and permitted to suck the nipple at intervals until the secretion of milk has been established. The colostrum exerts a laxative effect upon the bowels, and renders the administration of a purgative superfluous. There is seldom any necessity for the use of artificial foods pending the appearance of the milk; but in the case of very weakly infants, or where the secretion is unduly delayed, a little diluted and sterilized cow's milk may often be allowed with advantage. As soon as the secretion has become established, the infant should be applied to the breast at regular intervals of two hours during the day, each breast being emptied alternately; but between the hours of 11 p.m. and 4 a.m. it should be encouraged to sleep, so as to afford the mother the requisite amount of rest. This rule, however, is only applicable to healthy children, who exhibit a steady increase in weight. In feeble infants it may be advisable to allow three or four meals during the night. During the act of nursing, the child should be supported by the arm on the same side as the breast, while the mother bends slightly forwards, so as to allow the nipple to be easily grasped and retained in the infant's mouth, and steadies the breast with the fingers of her other hand. If the infant sucks feebly, slight pressure made upon the body of the gland will accelerate the flow of milk; but if the child withdraws the secretion too quickly, the flow can be retarded by judicious pressure upon the base of the nipple. In this manner the nurse can control the rapidity of the outflow at her will.

A child should never be forced to take the breast against its will, nor should it ever be permitted to retain the nipple in its mouth after it has exhausted the breast or ceased to suck. The habit which prevails among the poor of allowing an infant to sleep with the nipple in its mouth favours fermentation of the milk and ulceration of the breast. At the termination of each meal the breast should be cleansed with warm water and carefully dried with a soft towel; while the mouth of the child should be wiped out with a piece of wet linen, so as to remove any milk which may have remained concealed between the gums and the cheek. By the adoption of these precautions, the risk of sore nipples and of stomatitis is greatly diminished. After the second month the breast should be given every two hours and a half, and after the fourth month about every third hour. A healthy and vigorous infant usually empties the breast in ten to fifteen minutes, and at the end of the meal appears quite contented, and soon falls into a quiet sleep. Peevishness and wakefulness indicate that either the milk has disagreed with the child, or that it was insufficient in amount. The best test of the quality of the milk is to be found in the general appearance and condition of the suckling. At birth the average weight of a full-term infant is about seven pounds (six and a half to seven and a half pounds). During the first three days there is often some trifling degree of loss (four to eight ounces), owing to deficiency of milk; but after this a regular gain in weight takes place, so that about the eighth day the initial weight has been regained. From this time onward the growth of the body is rapid and continuous. At the end of the first month the weight has been augmented by one-third, by the fifth month it has been doubled, and at the end of the twelfth month trebled. After the end of the first year the growth is slower, so that it is not until the end of the sixth year that the weight is once more doubled. Careful observation of the weight of the body from week to week is, consequently, the surest guide to the general condition and progress of the infant.[1]

Refusal of the child to take the breast may arise from several causes. In many cases it is found that there exists either some local affection of the mouth or throat (stomatitis, thrush), which renders the process of sucking too painful to be endured, or some abnormal development of these parts (tongue-tie, cleft palate) prevents the child from exerting sufficient power of suction. In other cases, difficulty of respiration through the nose prevents the infant from imbibing milk in the ordinary manner. Thus, in many cases of syphilis, swelling of the nasal mucous membrane, or blocking of the orifice of the nares by scabs, obliges the child to breathe entirely through its mouth ; while in all acute diseases of the lungs and air-passages sucking is rendered almost impossible from the urgent dyspnœa which accompanies the complaint. On the other hand, retraction of the nipple may offer an almost insurmountable difficulty to the extraction of the milk, or the secretion itself, from the fact that it gives rise to abdominal pain, may prove distasteful to the child. In rare instances the presence of a post-pharyngeal abscess prevents the infant from swallowing the milk after it has extracted it from the breast.

---

[1] According to the observations of Pfeiffer, the average weight at the end of the first month is 8 lb. 5½ oz.; at the end of the second, 10 lb. 4 oz.; at the end of the third, 11 lb. 15 oz.; at the end of the fourth, 13 lb. 9¼ oz.; at the end of the fifth, 14 lb. 14½ oz.; at the end of the sixth, 16 lb. 3½ oz.; at the end of the seventh, 17 lb. 5 oz.; at the end of the eighth, 18 lb. 10 oz.; at the end of the ninth, 20 lb. 1 oz.; at the end of the tenth, 20 lb. 5½ oz.; at the end of the twelfth, 22 lb. 7 oz.

Contra-indications to Nursing.

Suckling must be prohibited whenever it appears certain that the process of nursing will involve danger to the mother or injury to the child. Thus, in women of a feeble or debilitated constitution, nursing often occasions severe exhaustion, and may pave the way to the inception of an incurable organic disease. Again, in cases of chronic phthisis, cancer, etc., maternal feeding is inadvisable, not only from the deleterious effects produced upon the mother, but also from the danger which sometimes accrues to the child by supplying it with insufficient nourishment or actively poisonous food. All acute febrile diseases in the mother likewise constitute a bar to the continuance of nursing, although, if the disorder only persists for a short time, it may be possible to employ the breast again. Syphilis in the mother does not as a rule necessitate weaning. Certain mental states, such as mania or melancholia, interfere with the process of nursing; and many highly nervous or emotional women are unable to continue to suckle their children, owing either to deficiency of the secretion or to some abnormal constitution of the milk (p. 23). The recurrence of menstruation or another pregnancy often act in an injurious manner; but the infant should not be permanently weaned unless the milk is found to continue to disagree after careful trial. Chronic dyspepsia and wasting of the infant is usually considered as an indication for weaning; but before this is undertaken, the quality and mode of administration of the milk should be carefully investigated, and the effect of 'mixed feeding,' if possible, be tried. Local affections of the breast—like abscess, cancer, retraction or ulceration of the nipple—may interfere with or negative its use, either by affecting the secretion, or rendering the extraction of the milk painful or difficult. Finally, it may be stated that after the age of twelve months the infant should be weaned as soon as possible.

Mixed Feeding.

It often happens that an infant at the breast fails to thrive properly owing to lack of sufficient nourishment, the milk being of average composition, but deficient in quantity. In such cases it is usually unwise to procure weaning, since a little human milk, if its quality is good, is better than none at all. The breast should only be administered two or three times a day, and in the intervals some other form of milk may be supplied to the child. In the case of infants less than six months old, cow's milk suitably diluted and sterilized, humanized milk (p. 40), or a cream mixture (p. 41), may be given; but at a more advanced age a little malted food (p. 54), whole-meal flour (R. 10), or meat broth, may be allowed once a day with advantage. By supplementing the natural food in this way, it is often possible to make use of the breast until the ninth or tenth month, and thus both to satisfy the ambition of the mother and assist the well-being of her child.

Weaning.

The exact period at which weaning should take place must be determined by the special circumstances of each case. As a rule, the question finds a natural settlement in a gradual failure of the mammary secretion, which necessitates supplementary feeding, and finally terminates in the child weaning itself. In other cases, the appearance of the teeth indicates that the infant is capable of digesting a certain amount of farinaceous food, the presence of six or eight incisors corresponding usually to the full development of the pancreatic secretion. Unless there exists some good reason to the contrary, an infant should never be weaned until after the sixth month. In cases where the milk continues to be secreted in sufficient quantity, there is no reason why nursing should not be continued until the end of the first year, provided always that the infant exhibits a steady increase in weight, and the mother suffers no injury to her health. If, however, the child ceases to gain in weight or the mother presents symptoms of exhaustion, the period of nursing should be curtailed, and weaning be procured. Under ordinary circumstances, a child should be weaned in a gradual manner, the breast being slowly withdrawn in favour of the bottle or spoon ; but it must never be discontinued entirely until the infant has grown used to artificial feeding, and shown itself capable of digesting the food supplied to it. If possible, weaning should not be performed during the hot months of the year, owing to the frequency with which a change of diet in the summer is followed by acute intestinal catarrh ; nor should it take place while the child is suffering from a temporary failure of its health. It is also wise not to wean a child while it is in the act of cutting a tooth.

After weaning has been completed, cow's milk should form the staple of the diet, being supplemented, if necessary, by custard puddings, mutton broth or jelly, or by some form of partially malted or pancreatized food (Diet, p. 57).

Diet in Lactation.

A woman who is nursing her child requires a plain, wholesome, and nutritious diet. The meals should be taken at regular intervals, and all excess of unduly rich and stimulating foods be excluded from the dietary. During the first week or two the mother should be encouraged to indulge freely in fluids, since a rich proteid diet at the commencement of lactation is apt to give rise to a milk containing an excess of solids. For this purpose, one or two glasses of milk during the day, or a cup of cocoa or gruel shortly before nursing, are of considerable value. Stimulants are seldom necessary; and the habit of ordering beer or stout to a nursing woman as a routine practice is to be deprecated. The effect of insufficient exercise upon the secretion of milk has already been noticed (p. 22). It is advisable, therefore, to recommend moderate exercise in the open air every day, care of course being taken to avoid fatigue. The bowels should be maintained in regular action, and for this purpose oatmeal-porridge at breakfast-time is to be recommended. Cathartics are to be avoided, owing to the deleterious influence they exert upon the mammary secretion; while the compound liquorice powder should be taken with caution, as it occasionally communicates laxative properties to the milk. If the milk is found to be *deficient in solids*, the proportion of fluids in the dietary should be diminished, the amount of exercise decreased, and the infant be applied to the breast at shorter intervals. If, on the other hand, the milk *contains an excess of solids*, the fluid portions of the diet must be increased, more exercise must be taken, and the intervals between the successive nursings prolonged. Since the amount of fat in the milk depends directly upon the quantity of proteid in the diet, the proportion of this ingredient can be readily adjusted by strict limitation of animal food.

The Wet-nurse.

Whenever a mother is unable to nurse her offspring, a wet-nurse should, if possible, be obtained. In the selection of a suitable woman for this important office, several points are deserving of consideration. The best nurses are those from twenty to twenty-five years of age, who have already borne one or two children. It is generally stated that women of a brunette type and vigorous constitution are better adapted for nursing than those of a more delicate build and fairer complexion. The researches of Becquerel and Vernois upon this subject, however, appear to indicate that fair women of delicate physique often secrete milk more closely allied to the standard than those of more robust development. In every case a physical examination must be undertaken, in order to ensure the absence of organic disease of the heart, lungs, liver, and kidneys, as well as such constitutional diseases as scrofula and syphilis. The catamenia should not have reappeared. As a rule, the milk of a woman who has already reached the third month of lactation is best adapted to the requirements of a young infant, since it has attained a uniform composition ; sufficient time has also elapsed to allow the condition of her own infant to be taken as a criterion of the value of her milk. If the child is plump and healthy, with a clear skin and a hearty appetite, and presents no signs of syphilis, it may be fairly argued that its mother's milk nearly approaches the standard both in quantity and composition. If, on the other hand, the infant exhibits any signs of wasting or skin disease, appears peevish and irritable, or ill satisfied with the breast, the woman is obviously ill adapted to take charge of another and younger child. The breasts of a healthy nurse should be small, firm, and pear-shaped, and present numerous blue veins ramifying over their surface. Gentle pressure should cause the milk to squirt out in fine jets, and the secretion under the microscope should present abundant globules of fat. If possible, a chemical analysis should be made, in order to determine the percentage value of its principal ingredients.

Despite the utmost care, it frequently happens that the milk of the woman selected immediately disagrees with the infant, and the nurse has to be changed in favour of another. Occasionally the milk of the nurse does not disagree until after the lapse of several weeks. In such cases it is generally found that the mammary secretion contains an abnormal amount of solids (proteids and fat), owing to insufficient exercise and an over-liberal diet. These defects are easily remedied by diminishing the quantity of animal food and increasing the daily exercise. In other instances, infantile dyspepsia arises from the recurrence of menstruation in the nurse, or from some temporary illness. In all cases the infant should be weighed each week, and in the event of it failing to gain in weight, the cause must be at once sought for and corrected.

ARTIFICIAL FEEDING.

Whenever circumstances render it impossible for an infant to be nourished by the breast, recourse must be had to some artificial form of food which closely resembles human milk in its physical characters and chemical composition. For this purpose the milk of one of the larger animals which suckle their young, such as the ass, mare, goat, and cow, is obviously the most suitable.

The milk of the ass and the mare most closely resemble the human secretion in composition, though they contain an inferior amount of both fat and proteid. The curd produced by their coagulation is also loose and flocculent, so that it is easily digested by the gastric juice. These advantages are, however, more than counterbalanced by the difficulty of obtaining them in sufficient quantity and at a reasonable price. Ass's milk also acts as an aperient unless previously boiled. The milk of the goat is much richer in caseinogen and fat than human milk, and often agrees well with infants; but it is expensive, and has a strong flavour, which sometimes renders it distasteful to a child. Cow's milk contains more proteid but less fat and sugar than woman's milk. It is obvious, therefore, that, whichever milk is adopted, it will require some degree of modification before it can be regarded as an adequate substitute for the breast; and since the necessary manipulation can be performed as easily with one kind as another, the choice of the milk must depend chiefly upon the relative supply and cheapness of the different varieties. Regarded in this way, cow's milk is infinitely superior to any other, and it is consequently the only one with which the problem of artificial feeding in infancy is immediately concerned.

Variations in Composition.

To a certain extent the secretion of milk in the cow is amenable to the same influences which affect that of a woman. Thus, it is found that the fluid varies in composition during the process of milking, and according to the length of time which is allowed to elapse between consecutive milkings. The relative percentage of its solid constituents likewise varies with the nature of the food, materials rich in carbohydrates, like carrots and beetroot, increasing the amount of sugar, while those containing an excess of proteids augment the quantity of caseinogen. When the refuse from breweries or distilleries is used for the purpose of feeding the cow, the milk becomes abundant in quantity, but poor in solids. Diseased potatoes or turnips utilized as fodder cause the milk to acquire an unpleasant taste and smell. As a rule, the finer the breed of the cow, the richer is its milk, so that the cattle of Jersey and Guernsey produce a milk containing an abundance of fat and proteid. The secretion is also susceptible to nervous influences affecting the cow, like fright or anger, and it may be often entirely suppressed by the administration of alum to the animal. Bacteriological examination has proved that the first portions of the milk drawn from the udder contain a considerable quantity of micro-organisms, which had gained an entrance to the ducts by way of the teat. After these have been washed out by the flow of milk, the secretion is found to be sterile. In diseased conditions of the udder (tuberculosis, infective mastitis) the milk usually contains pathogenic bacteria, and under all circumstances the fluid constitutes a most favourable medium for the development of those bacteria which gain access to it after its withdrawal.

Differences between Cow's Milk and Human Milk.

The milk of a cow differs from that of a woman in several important particulars. In the first place, it is much richer in proteids and salts, but poorer in sugar and, as a rule, also in fat. Recent investigations also tend to show that even the proteids and fat differ in chemical composition in the two forms of milk. In the second place, cow's milk as it is supplied to a household is invariably acid in reaction, and contains numerous bacteria, while human milk is alkaline and sterile. Thirdly, the total quantity of albuminoids present in cow's milk, which can be coagulated by means of acids, is almost four times as great as the non-coagulable; but in human milk the reverse obtains, there being an excess of non-coagulable albuminoids over the coagulable. Finally, cow's milk produces dense cheese-like curds, which offer almost insurmountable difficulties to the process of digestion. Human milk, on the other hand, produces a loose and flocculent precipitate of casein, which readily undergoes solution in the stomach and intestine. These main points of comparison between the two varieties of milk are well shown in the following table:

TABLE III.

	Human Milk (Leeds).	Cow's Milk (Leeds).
Reaction - - -	Alkaline	Acid
Specific gravity - -	1031	1029
Fat - - - - -	4·13	3·75
Lactose - - -	7	4·42
Proteids - - -	2·0	3·76
Ash - - - -	0·2	0·68
Bacteria - - -	Absent	Numerous
Coagulable albuminoids	Relatively small	Relatively great
Curd - - - -	Loose and digestible	Compact and indigestible

Care of the Milk.

In certain parts of the Continent and in America, where the subject of infant feeding is rightly regarded as a matter of importance, milk laboratories have been established for the purpose of modifying cow's milk in such a way as to render it similar in composition to human milk. At the farms connected with these establishments the cows are kept under strict and skilled supervision, and before being employed as milk-producers are proved to be free from any constitutional or local disease. The sheds in which they live are large, clean, and airy, and each separate stall is of sufficient size to permit of comparative freedom to the animal. The exercise-yard is large and dry, and pure water is provided for drinking purposes. The food is regulated on scientific principles, so as to produce a nutritious and digestible milk of even composition. The milkers are dressed in clean linen suits and caps, and carefully cleanse their hands and arms before commencing their task. The secretion is withdrawn with some force, and the udder is completely emptied on each occasion. The milk itself is received into glass-lined pails, and immediately carried to a shed close by, where it is poured through several thicknesses of sterilized gauze into a large chamber surrounded with ice. By this means the fluid is rapidly cooled, and the development of any bacteria which may have gained access to it is consequently retarded. Although these facilities for obtaining pure milk are at present wanting in this country, it is within the power of most people to select a dairy where the hygienic arrangements are good and the cows regularly inspected and properly cared for. As a rule, it is better to take a mixture of the milk from a herd than to endeavour to obtain that of a single cow, since in the former case the general composition of the fluid is less liable to vary. The milk should be delivered at the house as soon as possible, sealed up in clean glass bottles, which may be placed upon ice pending the necessary modification and sterilization. If these precautions cannot be ensured, the milk should be boiled immediately upon its receipt.

Modification of the Milk.

A glance at the foregoing table is sufficient to show that, in order to adapt the milk of the cow to the use of an infant, three points require attention: (1) The quantity of proteid matter must be reduced from 3·75 per cent. to 2 per cent.—*i.e.*, by almost one-half—the deficiency of sugar (2½ per cent.) and of fat (·8 per cent.) must be made good, and the reaction of the fluid converted from acid to alkaline; (2) the curd must, if possible, be prevented from forming in dense insoluble masses; (3) the fluid itself must be rendered free from bacteria before its administration to the child.[1]

1. *Simple Dilution.* — The simplest method of modifying cow's milk is by diluting it until its proteid constituents attain the same proportion as in human milk, and then to make up the deficiency in sugar by means of lactose. The diluent which is employed should be of such a kind as to prevent, as far as possible, the production of compact masses of curd in

---

[1] The researches of Budin and Chavanes indicate that many infants can digest undiluted cow's milk if carefully sterilized.

the stomach. With this object, various mucilaginous fluids, like barley, oatmeal, and rice-water (R. 1, 2, 3), or solutions of gelatine, isinglass (R. 4), and of malted flour (p. 54), have been recommended, upon the supposition that they can hinder the process of clotting in a mechanical manner, and thus bring about a fine subdivision of the curd. According to Rotch, the value of these fluids is purely imaginary, since a mixture of hot water and milk produces a finer form of curd than that which accrues from the use of any liquid holding particulate matter in suspension. Despite this assertion, however, it is found that well-strained barley-water is of greater value than plain water as a diluent for the milk, while it also appears to aid nutrition by the minute quantity of farinaceous material it contains. Although it is possible by simple dilution to reduce the percentage of proteids to the proper standard, they are not thereby rendered more digestible than before, so that it is usually necessary during the first month of infant life to dilute the milk with three or four times its volume of the attenuant solution. During the second month the milk may be administered along with an equal quantity of barley-water, and from the third to the sixth months the mixture may consist of two parts of milk to one of water. After the sixth month dilution is often unnecessary. In every case the food should be rendered alkaline by the addition of one-twentieth of its volume of lime-water (R. 5), and a quantity of lactose, varying from fifteen to sixty grains, be added to it, according to the proportion of milk it contains (Diet, I.).

Although this method of attenuation, combined with the addition of sugar, produces a mixture which is deficient in fat, and often in proteids also, it frequently agrees well with an infant, and is perhaps the most useful manner of prescribing milk in out-patient practice, where the more exact methods of feeding are out of the question.

2. '*Artificial Human Milk.*'—Various attempts have been made to imitate human milk by abstracting the excess of caseinogen from cow's milk, and supplying the lack of fat and sugar by the addition of cream and lactose. The method proposed by Frankland for the production of an artificial human milk is as follows: 'Allow one-third of a pint of new milk to stand twelve hours; remove the cream, and mix it with two-thirds of a pint of perfectly fresh cow's milk. Take the blue milk, from which the cream was removed, and put a piece of rennet about an inch square (fluid rennet may be employed) into it. Keep the vessel containing it in a warm place until the milk is fully curdled—an operation requiring five to fifteen minutes, according to the activity of the rennet, which should be removed as soon as the curdling commences. Break up the curd repeatedly with a fork, and carefully separate the whole of the whey, which should then be rapidly heated to boiling in a small tin pan placed over a spirit or gas lamp. During the heating a further quantity of casein, technically called "fleetings," separates, and must be removed by straining through clean muslin. Now dissolve 110 grains of powdered sugar of milk in the hot whey, and mix it with the two-thirds of a pint of new milk, to which the cream from the other fluid has already been added as already described. The artificial milk should be used within twelve hours of its preparation; and it is almost needless to add

that all the vessels employed in its manufacture should be kept scrupulously clean.'

By another method the cream is separated from a pint of milk, and the casein of one-half of the skimmed milk coagulated by means of rennet, and strained off. To this whey, after boiling, the cream which was removed, and the rest of the skimmed milk, is added, and the whole sterilized. Such a mixture contains on an average about 2 per cent. of proteids, 4·5 per cent. of fat, 5 per cent. of sugar, and ·66 per cent. of salts.

Gaertner has employed the centrifugal cream-separator to modify cow's milk in such a way as to provide a mixture (*Fettmilch*) closely resembling human milk in the amount of fat and proteids it contains. Before being placed in the cistern of the machine, the milk is diluted with an equal quantity of water, and warmed to the temperature of the body. The apparatus revolves at such a rate that the quantity of fluid delivered by the two tubes is equal in amount. The fluid which flows from the tube connected with the centre of the revolving funnel contains almost all the fat, along with one-half of the proteids, sugar, and salts, so that it is only necessary to add to it about 4 per cent. of lactose to make it closely resemble human milk in composition.

3. '*Cream Mixtures.*'—Biedert and Meigs having shown that it was possible to imitate human milk in a satisfactory manner by an artificial combination of its principal ingredients along with cow's milk, various cream mixtures have been devised for the purposes of infant feeding. One of the best mixtures of its kind is that recommended by Ashby : A glass bottle, provided with a stopcock at its lower part, is gradu-

ated to fifteen and thirty fluid ounces respectively, and, after being filled to the upper mark with fresh milk, has its mouth plugged with cotton-wool. After standing for two hours at the ordinary temperature of a room, a certain amount of cream will be found to have risen to the top. The stopcock is now opened, and the lower half of the fluid drained off, and replaced by a 7 per cent. solution of milk-sugar (made by dissolving two measured ounces of milk-sugar in a sufficient quantity of warm water to make fifteen ounces in all) ; the mixture is then sterilized, and afterwards rapidly cooled. If the milk used is of good average quality, the mixture will have somewhat of the following composition : Fat, 2·6 per cent. to 3 per cent. ; lactose, 5 per cent. to 6 per cent. ; proteids, 1·7 per cent. to 2 per cent. One-twentieth of its volume of lime-water should be added before its administration to the infant. If it be desired to increase the percentage of fat in the mixture, the milk must be allowed to stand for a longer period than two hours.

By means of a centrifugal apparatus, milk can be deprived almost entirely of its cream within a short time, and both it and the separated milk may be utilized in the manufacture of feeding mixtures. Rotch recommends that the cream should be thus separated from a pint of milk, which is then divided into halves. To one-half of the separated milk the entire quantity of cream is added, and the mixture made up to a pint by the addition of an 8 per cent. solution of lactose. The same writer gives a valuable table for the manufacture of cream mixtures, each of which corresponds fairly accurately to a definite formula (see Appendix R.). It will be observed that in each of these three modifications of cow's milk the caseinogen remains unaltered in character, and may, therefore, still prove difficult of digestion. In order to obviate this, it may be necessary to peptonize the mixture before its administration (p. 53).

SCHEME OF DIET FOR THE FIRST SIX MONTHS.[1]

For First Week.

Cream	2 fl. drs.	or	Cow's Milk	½ fl. oz.
Whey	3 " "		Milk-sugar	15 grs.
Milk-sugar	10 grs.		Barley-water	½ fl. oz.
Water	3 fl. drs.			

Every 2 hours (night and day).

For the Second to the Fourth Week.

Cream	2 fl. drs.	or	Cow's Milk	6 fl. drs.
Cow's Milk	½ fl. oz.		Milk-sugar	15 grs.
Milk-sugar	15 grs.		Barley-water	5 fl. drs.
Water	1 fl. oz.			

Every 2 hours (4 a.m. to 10 p.m.).

For the Third Month.

Cream	3 fl. drs.	or	Cow's Milk	12 fl. drs.
Cow's Milk	12 " "		Cream	3 " "
Milk-sugar	28 grs.		Sugar of Milk	30 grs.
Water	1 fl. oz.		Barley-water	12 fl. drs.

Every 2½ hours (5 a.m. to 11 p.m.)

For the Fourth Month.

Cream	3½ fl. drs.	or	Cow's Milk	2 fl. oz.
Cow's Milk	12 fl. [illegible]		Cream	4 fl. drs.
Sugar of Milk	40 grs.		Sugar of Milk	45 grs.
Water	2 fl. oz.		Barley-water	16 fl. drs.

Every 2½ hours (5 a.m. to 11 p.m.).

For the Fifth Month.

Cream	4 fl. drs.	or	Cow's Milk	18 fl. drs.
Cow's Milk	2 fl. oz.		Cream	5 " "
Sugar of Milk	50 grs.		Sugar of Milk	60 grs.
Water	1½ fl. oz.		Barley-water	2 fl. oz.

Every 3 hours (5 a.m. to 11 p.m.).

---

[1] Modified from Angel Money and Louis Starr.

For the Sixth Month.

Cream	- 4 fl. drs.	or	Cow's Milk	- 20 fl. drs.
Cow's Milk	- 20 „ „		Cream	- 5 „ „
Sugar of Milk	- 60 grs.		Sugar of Milk	- 60 grs.
Water	- 1 fl. oz.		Barley-water	- 1½ fl. oz.

Every 3 hours (5 a.m. to 11 p.m.).

Preserved Milks.

The difficulty of procuring fresh milk in large towns, and of keeping it for many hours without the risk of decomposition, has led to the introduction of certain methods of preserving milk. This can be effected in three ways: (1) By the addition of such substances as boracic acid, formalin, etc., which hinder fermentation; (2) by partial evaporation of the milk (condensation), with or without the addition of cane-sugar; (3) by evaporation to dryness. The first method, being quite unsuitable, need not be discussed.

Condensed Milks are of two kinds: 'sweet' and 'unsweetened.' They are both made by evaporating fresh milk to about one-third of its bulk; but in the sweet variety about 20 per cent. of cane-sugar is added, in order still further to hinder the process of decomposition. Owing to the portable form of the food, the ease with which it can be prepared, and the length of time it will keep, the sweet condensed milk is very largely employed by the poorer classes for the purposes of infant feeding. But even if it be allowed that the curd of condensed milk is more easily digested than that of fresh milk, the apparent advantages possessed by it are far more than counterbalanced by the ill effects which usually accrue from the habitual employment of these foods. In the first place, a large proportion of the so-called condensed milks that are put on the market are found on analysis to have been prepared from 'skimmed milk,' and show an average of only 0·72 per cent. of fat. Others, again, are made from 'separated milk' to which a small amount of fresh milk has been added, and show an average of only 3·14 per cent. of fat; while samples of genuine condensed 'full-cream milk,' such as the 'Milkmaid' brand, have yielded from 10 per cent. to 12 per cent. of fat on analysis (Parkes). It is obvious, therefore, that, unless due care be taken in the selection of a reliable brand, it is quite possible to supply an infant with 'condensed separated milk' almost free from fat, and consequently devoid of its most nutritious principle.

Again, unless the milk is carefully diluted with a due amount of water, there is always the danger that its percentage of proteids will either overtax the powers of digestion, or be so much reduced as to cause the solution to lack a sufficiency of nourishment. Owing to the large amount of sugar it contains, the sweet milk should be diluted with fifteen or twenty times its weight of water (half a teaspoonful to three tablespoonfuls) before being used for an infant less than a week old. Such a solution is deficient both in proteids and fat, so that a small quantity of meat-juice and fresh cream will have to be added to it. After the second month the milk should be diluted with about ten parts of water. The unsweetened ('Viking' brand) variety should at first be diluted in the proportion of one to six, but at a later period a one in four solution may be employed.

The rapidity with which cane-sugar undergoes fermentation in the infant's stomach is usually considered a strong argument against the employment of sweet condensed milk as a substitute for maternal feeding; and there can be no doubt that the careless administration of strong sweet milk is a frequent cause of intestinal fermentation in young infants. It is also found that children who have been fed by means of condensed milk, though often very fat, are usually pale and flabby, and prone to develop rickets or to fall victims to intestinal catarrh. Occasionally it would appear that a kind of scurvy ensues from the use of preserved milks, indicating that the antiscorbutic properties of the fresh milk may possibly be destroyed in the process of condensation. Finally, the contention that these preserved milks are sterile has been contested of late years, and bacteria have been cultivated from freshly-opened tins. For these several reasons, condensed milk cannot be considered as either a safe or efficient substitute for carefully modified fresh milk, and its use should, therefore, be reserved for those cases where a temporary failure of the infant's powers of digestion renders it impossible to prescribe fresh milk with success.

The following table shows the composition of the two forms of condensed milk in their undiluted condition, and also in that most suitable for administration to an infant (Leeds):

TABLE IV.

	Human Milk.	Sweet Condensed Milk.	Diluted 1 to 8 (by weight).
Fat - -	4·13	12·10	1·51
Lactose - -	7·0	16·62	2·06
Cane-sugar -	—	22·26	2·78
Proteids -	2·0	16·07	2·01
Ash - -	0·2	2·61	0·32
Total Solids -	13·33	69·66	8·68

	Unsweetened Condensed Milk.	Diluted 1 to 6 (by weight).
Fat - -	9·9	1·65
Lactose - -	13·3	2·2
Proteids - -	8·9	1·5
Ash - -	1·9	0·16
Total Solids -	34·0	5·51

Dried Milks are often valuable substitutes for fresh milk in cases where the latter cannot be procured or proves difficult of digestion. The best form is that prepared by Allen and Hanbury (No. 1), and must be mixed with a measured quantity of water before being given. The other dried-milk foods prepared by the same firm contain malt.

Size and Frequency of the Meals.

The capacity of the stomach at birth is very small (one ounce), while the process of digestion is comparatively rapid. An infant consequently requires small, but frequent, meals. From a series of observations conducted upon infants less than a month old, Ssnitkin came to the conclusion that the size of the stomach varies directly with the weight of the body; and he has formulated a law to the effect that, whereas at birth a child requires an amount of food equal to one one-hundredth part of its body-weight, an additional gramme of milk must be added to each meal for each subsequent day of life until the end of the first month. Thus, if a child at birth is found to weigh three thousand grammes, it will require thirty grammes of milk per meal (*i.e.*, about one fluid ounce), while on the fifteenth day it will need thirty plus fifteen grammes, or about one and a half fluid ounces.

It may be accepted as a general rule that during the first week of life an infant should be given one fluid ounce of food every two hours, day and night, and for the rest of the first month about one and a half ounces at the same intervals of time. During the second month from two to three ounces may be given every two hours and a half during the day, and once or twice during the night. In the third and fourth months about four ounces should be allowed every two hours and a half; but the child must be encouraged to sleep continuously between the hours of 11 p.m. and 5 a.m. During the fourth month four and a half ounces should be allowed at each meal, and during the fifth and sixth months five ounces every three hours. The general rules of feeding during the first year are well expressed in the following table (Rotch):

TABLE V.

Age.	Interval (hours).	Amount at each Feeding (ounces).	Total Amount in 24 Hours (ounces).
1 week -	2	1	10
2 weeks -	2	1½	15
4 " -	2	2½	22½
6 " -	2½	3	24
8 " -	2½	3¼	28
3 months -	2½	4	28
4 " -	2½	4½	31½
5 " -	3	5½	33
6 " -	3	5¾	34½
7 " -	3	6¼	37½
8 " -	3	7	42
9 " -	3	7	42
10 " -	3	8½	42½
11 " -	3	8¾	43¼
12 " -	3	9	45

In all cases it is better to prefer the risk of underfeeding rather than of overfeeding a child, since the effects of the former are easily recognised and soon remedied, while the disorders of digestion which arise from overcharging the stomach with food often escape detection until they have produced a serious effect upon the general nutrition.

Sterilization of the Food.

Since milk becomes readily infected by exposure to the air or by admixture with non-sterile fluids, it is necessary to destroy any micro-organisms it may contain before its administration to the infant. This can be most easily accomplished by the application of heat; but the exact temperature which it is necessary to procure in order to effect sterilization varies with the nature and number of bacteria present. Most of the pathogenic organisms (tubercle, typhoid, diphtheria) can be destroyed by exposure to a temperature of 70° C. for half an hour; but the spore-bearing species, which decompose milk with the production of poisonous peptones and albumoses, require a much higher temperature to ensure their destruction. As a rule, the staler the milk, the greater the number of its bacteria, and the greater the chance of its containing spores and organic poisons. It follows, therefore, that while fresh milk requires less heating than stale, no amount of sterilization can convert an actively poisonous milk into a nutritious food.

1. *Pasteurization.*—In this process the milk is never heated to a temperature higher than 70° C.; it is, therefore, only to be used where the milk can be

obtained in an absolutely fresh state. The employment of a comparatively low temperature has the advantage of not producing any unpleasant taste or smell in the milk, nor is it attended by the formation of a permanent scum on the surface of the fluid, which results from the coagulation of the lactalbumin. The apparatus required consists of: (1) A glass bottle of moderate size, with a wide mouth; (2) a metal vessel or 'boiler' fitted with a lid, through an aperture in which a thermometer can be inserted; (3) a wire stand for the support of the bottle; (4) a long thermometer. The bottle having been filled with milk, its mouth is plugged with cotton-wool, and it is placed upon the wire stand inside the boiler. Water is poured into the latter so as to surround the bottle, the lid is put on, the thermometer inserted, and the whole placed upon a stove until the water has been raised to the temperature of 70° C. When this has been attained, the boiler is removed from the fire and covered with a cosy for half an hour. The bottle is then withdrawn and immersed in cold water so as to rapidly reduce the temperature of its contents, and kept in a cool place until required.

2. *Sterilization.*—In this process the milk is maintained at the temperature of boiling water for about forty minutes. It is to be preferred to 'pasteurization' whenever any doubt exists as to the freshness and purity (non-adulteration) of the milk, and is consequently best adapted to the requirements of households situated in large towns. The disadvantages are that, from the high temperature employed, the milk acquires a boiled taste and smell, the lactalbumin is coagulated, while the fat emulsion is partially destroyed,

and gives rise to large drops of butter, which float on the surface of the fluid. It is also possible that the antiscorbutic properties of fresh milk are injured by the severe heat. On the other hand, the precipitation of some of the calcium salts which occurs renders the caseinogen less easy of coagulation, and in this way may favour its digestion in the stomach ; while any organic matter which may be present is destroyed by the high temperature.

The *Sterilizer* consists of a large sheet-tin vessel or 'steamer' provided with a lid, and of sufficient size to accommodate eight feeding-bottles. The amount of food required for a meal is put into each bottle, the mouth of which is then plugged with cotton-wool, and covered with an indiarubber cap. The bottles are arranged upon a wire support inside the steamer, and surrounded with water. Heat is applied to the apparatus, and the water kept boiling for forty-five minutes. The bottles are then removed and kept in a cool place. If a proper sterilizer cannot be obtained, the feeding-bottle may be placed in a saucepan of boiling water, and kept on the fire for about forty minutes, care being taken to prevent the bottle from coming into contact with the bottom of the vessel.

The Feeding Apparatus.

This should always be of the simplest construction possible. The bottles should be oval in shape, wide-mouthed, and capable of holding about ten fluid ounces, and, if possible, be graduated in half-ounces. All forms of corks and tubing must be avoided, owing to the difficulty of keeping them clean. The teats should be made of soft indiarubber, and large enough to permit of being turned inside out. It is often necessary to try several teats before one can be found to suit the infant's taste. A fresh teat should be provided once or twice a week. It is best to keep a large number of bottles constantly in use, so that the food for the whole day can be sterilized at one time. At meal-time the bottle containing the proper amount of sterilized milk is warmed by means of hot water, its cotton-wool plug withdrawn, and a clean teat drawn over the mouth. As soon as it has been emptied, the bottle should be rinsed out with water and then boiled, after which it can be again filled with milk and put into the sterilizer. The teat is turned inside out, and well scrubbed with a clean nail-brush; it may then be immersed in a dilute solution of caustic soda until required. If these simple precautions are taken, the chance of gastric infection from dirty tubes or sour bottles is rendered impossible.

Intolerance of Milk.

However carefully the food may be prepared, a certain number of infants will always be found who, during the first few weeks of life, are unable to digest cow's milk. In such cases an attempt must be made either to render the caseinogen of the milk more easy of digestion, or to substitute for it some other form of proteid matter, which does not offer the same difficulties to solution by the gastric and intestinal juices.

1. *Strippings and Whey.*—'Strippings' is the term applied to the additional supply of milk which can be withdrawn after the ordinary amount has been taken

from the udder. It contains a larger percentage of fat than ordinary milk, but less proteids, so that it can sometimes be digested by delicate children when suitably diluted with alkalinized barley-water. It is chiefly of value in mild cases of milk indigestion, where humanized milk cannot be properly made.

When all the caseinogen has been removed from fresh milk by means of rennet, the resultant fluid, or 'whey,' is found to contain most of the fat and salts of the original milk, along with lactalbumin and 'whey proteid.' Its employment is chiefly indicated in early infancy, before the establishment of the breast secretion, and in cases of feeble digestion, where the use of modified cow's milk is followed by vomiting and the passage of curd. As the fluid is deficient both in fat and albumin, a little cream, with meat-juice or white of egg, should be added to it, in order to increase its nutritive value.

2. *Predigested Milk.*—It is always possible to render cow's milk easy of digestion by submitting it to the influence of the pancreatic ferment before its administration to the infant. This method can either be carried out to its full extent, and the whole of the caseinogen be converted into peptones, or it may be stopped halfway, so as to leave a certain amount of undigested material, upon which the digestive fluids may act. Peptonized milk is of considerable value in the treatment of indigestion, but it must always be regarded in the light of a temporary substitute for fresh milk, and should never be persisted in for any length of time. Infants who are nourished solely upon predigested foods soon lose their natural powers of digestion, and are apt to suffer from anæmia and

rickets, and not infrequently present symptoms allied to scurvy.

Peptonized milk is best prepared in the following manner: A pint of milk is diluted with a quarter of a pint of water, and mixed with two teaspoonfuls of the Liquor Pancreaticus (Benger) and about twenty grains of bicarbonate of soda. The vessel containing it is then placed in the sterilizer, and the temperature of the water gradually raised to 60° C., and then more quickly to the boiling-point. The chief point aimed at is to prevent the development of the bitter taste which arises from over-peptonization. This can be avoided by carefully regulating the dose of the pancreatic solution and the time occupied by the process. If a sterilizer is not to hand, the mixture should be kept in a covered jug in a warm room for an hour or more, and afterwards boiled. This method of peptonization can of course be applied to any form of milk or cream mixture.

3. *Malted Foods.*—It is occasionally advisable to supplement the diet of an infant less than six months old by means of gruel which has been previously digested with malt. In no case, however, should undigested farinaceous matter be administered to a child before the seventh month. The employment of ground malt, as proposed by Liebig, is attended by so many difficulties that Sir William Roberts has proposed the substitution of an infusion of malt (R. 16) for the dry material, and recommends that it should be used in the following way: 'A suitable gruel is prepared from wheat, or from oatmeal, groats, pearl-barley, arrowroot, or any other farina. The gruel may be made with water alone, or, as is more usual, with the

addition of milk or some kind of meat broth. In either case the gruel should be well boiled and strained to separate the lumps. When the gruel is cold, or at least sufficiently cool to be tolerated in the mouth, the malt infusion is added. One tablespoonful (well mixed therewith) is sufficient to digest half a pint of gruel. The action is very rapid; in a few minutes the gruel becomes thin from the conversion of the starch. When this point is reached the food is ready for use. The only precaution to be observed in the process is to make sure that the gruel is at least sufficiently cool to be borne in the mouth before the malt infusion is added. It is not of the least consequence if the temperature be below this point, for the transformation goes on just as well, though not so rapidly, when the gruel is cold as when it is warm, whereas too high a temperature endangers the activity of the ferment, which is rendered inert at 170° F. The product of the action of malt on starch is maltose, and maltose has very little sweetening power. This is the reason why gruel thus digested suffers little change of taste—so little, indeed, that the addition of it to milk or broth produces hardly any appreciable alteration of flavour.' Malt extracts are less suitable than the infusion, since they communicate a brown colour to the food, and are much more expensive. Among the patent malted foods, those prepared by Mellin, Savory and Moore, and Allen and Hanbury, are the most reliable substitutes for the home-made article.

Diet from the Sixth to the Twelfth Month.

The appearance of the teeth usually coincides in point of time with the development of the diastatic functions of the pancreas and salivary glands. It may therefore be regarded as an indication that the infant will soon be able to digest starchy foods. In all cases the farinaceous material must be well boiled before being given, and the amount should be carefully regulated according to the digestive capabilities of the child. Once or twice a day a teaspoonful of well-boiled oatmeal, whole-meal flour (R. 10), or cooked maize, may be made into a gruel with milk, or a teaspoonful of bread or barley jelly (R. 11) may be employed instead. Milk must remain the staple diet, and at least one and a half to two pints should be allowed every day. If signs of indigestion follow the use of farinaceous food, or the infant ceases to gain in weight, the gruel should be predigested with malt, or a little malt extract added to the porridge. After the twelfth month the bottle should be omitted in favour of spoon-feeding.

Diet after the First Year.

If the child continues to increase in weight, and appears strong and healthy, bread-and-milk may be allowed once a day after the end of the first year, or a lightly-boiled yolk of egg, a custard pudding, or some stale breadcrumbs soaked in gravy, may be given with the mid-day meal. After the appearance of the first set of double teeth, a little finely-chopped fish, chicken, or meat, can be given along with gravy and potato. A small quantity of stewed fruit is also a useful adjunct to the meals. The general schemes of diet at different ages are shown in the following tables :

SCHEME OF DIET FOR AN INFANT FROM SIX TO TWELVE MONTHS OLD.

First Meal at 7 *a.m.*—A teacupful of diluted alkaline milk, humanized milk, or cream mixture.

Second Meal at 10.30 *a.m.*—A teacupful of one of the aforementioned, with a teaspoonful of malted food (Mellin's), or bread jelly, or two teaspoonfuls of barley jelly.

Third Meal at 2 *p.m.*—The same.

Fourth Meal at 6 *p.m.*—The same.

Fifth Meal at 9.30 *to* 10 *p.m.*—Same as the first meal.

After the seventh month a teaspoonful of whole-meal flour may be given instead of the malted food at the second or third meals.

A little fine oatmeal-porridge may be occasionally allowed with the first meal.

After the ninth month the yolk of one lightly-boiled egg may be given with the third meal; or a cupful of veal, chicken, mutton, or beef broth, be allowed, along with stale breadcrumbs.

SCHEME OF DIET FOR AN INFANT FROM TWELVE TO EIGHTEEN MONTHS OLD.

First Meal at 7 *a.m.*—A breakfastcupful of milk, with a slice of thin bread-and-butter or a rusk; or eight ounces of hot milk, with half a slice of stale bread well soaked in it; or a breakfastcupful of milk, with the yolk of a lightly-boiled egg and a slice of bread-and-butter; or a teaspoonful of Quaker Oats, made with milk and salt.

Second Meal at 10 *a.m.*—A breakfastcupful of milk, with a thin slice of bread-and-butter, a rusk, or a plain biscuit.

Third Meal at 1 *p.m.*—A teacupful of good beef-tea, with a slice of stale bread; or a little well-steamed mealy potato, mashed up with eight tablespoonfuls of gravy, and a little tapioca or sago pudding; or the yolk of a lightly-boiled egg, with bread-and-butter and a little junket.

Fourth Meal at 4.30 *p.m.*—A breakfastcupful of milk, with a rusk, dry biscuit, or slice of stale bread-and-butter.

Fifth Meal at 10 *p.m.*—A cupful of milk or a small basin of bread-and-milk.

The child should take between one and a half and two pints of milk every day.

The constitution of the meals should be varied as much as possible from day to day.

A little stewed fruit may be given once or twice a week with the mid-day meal.

After the *fourteenth month* the last meal at night may usually be dispensed with.

SCHEME OF DIET FOR A CHILD FROM EIGHTEEN MONTHS TO TWO YEARS OLD.

First Meal at 7.30 *a.m.*—Eight to ten ounces of milk, with two slices of bread-and-butter; or a basin of bread-and-milk; or a tablespoonful of Quaker Oats, made with milk and cream; or a breakfastcupful of milk, with the yolk of a lightly-boiled egg and a slice of bread-and-butter.

Second Meal at 10.30 *a.m.*—A breakfastcupful of milk, with a rusk, a plain biscuit, or a slice of bread-and-butter, with a little treacle, sugar, or marmalade.

Third Meal at 1.30 *p.m.*—A cupful of beef-tea, or mutton, veal, or chicken broth, with a slice of stale bread, soft toast, or rusk, and a custard pudding; or well-mashed potato, with gravy and bread-crumbs, and a little junket, tapioca, or rice pudding made with milk; or a small quantity of finely-minced or pounded mutton-chop, mixed with gravy and potato; or a lightly-boiled egg, with bread-and-butter and pudding.

Fourth Meal at 5 *p.m.*—The yolk of a lightly-boiled egg, with bread-and-butter and cocoatina (half a teaspoonful to six ounces of milk); or a basin of bread-and-milk; or a cupful of milk, with two slices of bread-and-butter and a little treacle.

Fifth Meal at 8 *p.m.*—A teacupful of milk (if necessary).

A little stewed celery, or well-cooked asparagus or cauliflower, may be given occasionally with the mid-day meal.

The diet should be varied as much as possible from day to day.

When the child has cut its first set of double teeth, a small quantity of boiled sole or cod may be given once a day, alternated with finely-minced boiled fowl or underdone mutton-chop.

CHAPTER III.

THE DYSPEPTIC CONDITIONS OF INFANCY

(VOMITING, SIMPLE DIARRHŒA, COLIC, ETC.).

ETIOLOGY.

FUNCTIONAL disturbances of the digestive organs occurring during the period of infancy are invariably due to some error of diet, and are therefore more often encountered in hand-fed children than in those nourished exclusively upon the breast.

Among one hundred consecutive cases of dyspepsia in infants less than twelve months old which came under my care at the Evelina Hospital, nine were found to have been fed entirely upon the breast, while the remaining ninety-one were in the habit of receiving either cow's milk or some form of farinaceous food. These figures, while they express fairly accurately the relative frequency of dyspepsia among sucklings and hand-fed infants in the poorer classes of society, also afford the material for a discussion of the various causes which give rise to indigestion in early life.

Breast-fed Children.—The natural law that the milk of every woman is especially adapted to the digestive capacities of her own child finds few exceptions in practice. Occasionally, however, cases are encountered where an infant exhibits a remarkable intolerance of its mother's milk, although it is able to digest with ease the milk of other women. This is often ascribed to some idiosyncrasy on the part of the child, but it is much more probable that in such cases the mammary secretion of the mother presents some chemical peculiarity which renders it difficult of digestion. A more frequent cause of indigestion in sucklings is to be found in the deterioration of the milk which arises from acute illness of the mother (p. 23). Thus, in three out of the nine cases to which allusion has been made the mother was found to be suffering from either diarrhœa, tonsillitis, or influenza, and in each instance it was positively ascertained that the first symptoms of illness in the child had ensued immediately after the mother had become unwell. In other cases, again, a sudden and violent emotion appears to induce some change in the chemical composition of the milk which renders it difficult of digestion by the infant, possibly by increasing the percentage of its proteids.

A good example of this recently came under my notice in the person of a young lady who, whilst nursing her first child, had a violent quarrel with a near relative. The next time she fed the infant it was seized with vomiting, and rejected several hard masses of curd, like those produced by the coagulation of cow's milk, and this recurred after each subsequent meal. These symptoms rapidly subsided as soon as the bottle was substituted for the breast, and did not return when the nursing was resumed a week later. But in many cases of dyspepsia in sucklings it is not so much the quality of the milk as the manner in which it is administered that is immediately responsible for the digestive disorder. It has already been observed

(p. 3) that the capacity of the infantile stomach is strictly limited, and that at least two hours are required for the adequate performance of the processes of digestion. In spite of this elementary fact, many persons habitually overfeed their children, either by giving them the breast too frequently, or by allowing them to imbibe too much milk at a time. Among the lower classes the breast is usually regarded in the light of a baby-comforter, and the nipple is at once thrust into the child's mouth should it cry or show the least signs of peevishness. It follows that under these circumstances the work of the stomach is never finished, and digestion consequently becomes a continuous, instead of an intermittent, process. The need of physiological rest thus engendered soon makes itself apparent, for the organ ceases to respond to the incessant calls upon its secretory functions, its muscular power fails, and the milk stagnates and undergoes fermentation. The unfortunate child, tormented with flatulence and colic, endeavours to express its suffering by screams and facial contortions, which, being interpreted by its mother as indicative either of hunger or temper, are promptly quelled by a fresh application of the breast; so that the mischief, when once begun, proceeds in a progressive manner, until life is placed in jeopardy by the sudden supervention of acute gastro-intestinal catarrh. Among the better classes the same result is sometimes brought about in a different manner. The regular administration of the breast being often incompatible with the due performance of the various social duties, the infant has perforce to receive its nourishment at odd times, and being often extremely hungry, is induced to swallow more milk than it has the power to digest. This gives rise to dilatation of the stomach and fermentation of the food. Another point of interest in this connection is the fact that irregularity of nursing adversely affects the quality of the mammary secretion; too frequent suckling renders the milk indigestible by increasing the percentage of its solids, while infrequent feeding is productive of a watery and innutritious fluid (p. 22).

Grave disease in the mother, such as phthisis, anæmia, carcinoma, and Bright's disease, is often accompanied by a deterioration in the quality of the milk, which renders it unsuitable for her child. The recurrence of menstruation during the period of lactation is also apt to be accompanied by temporary changes in the mammary secretion which are provocative of indigestion; while a fresh pregnancy not infrequently necessitates premature weaning for a similar reason (p. 23). Another factor of importance in the causation of dyspepsia is to be found in the habit which exists among certain women of allowing the infant to sleep with the nipple in its mouth. The milk which stagnates in the buccal cavity rapidly ferments, and, being swallowed by the child, infects the stomach and leads to fermentation of the milk of the next meal. Finally, it may be noticed that painful abdominal symptoms occasionally arise in infants at the breast owing to the accidental imbibition of poisonous substances along with the milk. Thus, certain drugs, like iodide of potassium, morphine, lead, and mercury, when administered to a nursing woman, are partially eliminated in the secretion of the breast, and, being ingested by the infant, give rise to phenomena of a toxic nature. In like manner the use of cosmetics by the mother has been known to be followed by symptoms of poisoning in the infant; while the use of lead or other medicinal applications to the nipple is occasionally responsible for the appearance of alarming and mysterious symptoms in the suckling.

When dyspepsia occurs in infants who are suckled by wet-nurses, the cause is usually to be found in an abnormally rich condition of the milk (p. 33). As the period of lactation advances, the percentage of caseinogen in the mammary secretion is often augmented, so that, unless especial care be taken to select a woman whose own offspring is about the same age as her foster-child, her milk is very apt to disagree. Another cause of milk dyspepsia in nurslings arises from overfeeding on the part of the nurse; for it has been shown by analysis that the effect of a rich animal diet is to increase the percentage of proteids in the milk, and thus to render it unsuitable for digestion in early infancy (p. 23).

Hand-fed Children.—The extreme liability to dyspepsia exhibited by children fed in an artificial manner appears to arise from two principal causes. In the first place, the foods themselves are ill adapted to digestion, and, in the second, they are prepared in such a way as to favour the entrance of micro-organisms. Among the ninety-one cases of dyspepsia occurring in hand-fed children, it was found on inquiry that in fifteen the food consisted entirely of cow's milk, in fifty-one sweet condensed milk was employed, while in twenty-five some form of farinaceous material constituted the staple of the diet. The chief cause of the dyspepsia which ensues from the employment of cow's milk is to be found in the excess of proteids it contains and the insoluble character of its curd. In large towns the milk is also as a rule adulterated, and is not infrequently sour before it is given to the child. The feeding apparatus is seldom cleaned, and the long rubber tube which connects it with the teat is often half choked with dirt and fermenting curd. It is hardly a matter of wonder, therefore, that among the poor the use of cow's milk is so often accompanied by bad results.

The frequency with which indigestion follows the use of condensed milk arises mainly from the careless manner in which it is administered and from the excessive quantity of fermentable sugar it contains. As a rule, the infant is supplied with a solution which is far too strong, and its stomach is consequently unable to digest with sufficient rapidity the excess of casein presented to it. Fermentation consequently occurs, and the stomach and bowels become distended with gas, while the mucous membrane of the alimentary tract is irritated by the various organic acids which are produced. Farinaceous foods are particularly unsuitable to the requirements of the system during the first three months of life, owing to the absence of the diastatic ferment from the saliva and the comparative inefficiency of that in the pancreatic juice. It is obvious, therefore, that infants who are supplied with an excess of starch, without at the same time possessing the power to assimilate it, will not only suffer from malnutrition, but are likewise exposed to the dangers which arise from its fermentation in the digestive tract.

The other causes of dyspepsia in hand-fed children are similar to those already described in the case of infants at the breast. Occasionally, the food becomes accidentally contaminated with vegetable or mineral poisons derived either from the water employed in its preparation or from some part of the feeding apparatus. Thus, cases have been described where urgent symptoms followed the use of milk that had been in contact with the leaden stopper of a bottle,[1] while in others symptoms of arsenical or lead poisoning were traced to the shot employed for the purpose of cleansing the feeding-apparatus.[2] At certain seasons of the year, notably in the autumn, acute dyspepsia often assumes an epidemic character, while many infants develope symptoms of the disorder whenever they are exposed to cold or wet. In these latter cases, however, the phenomena of indigestion are probably dependent upon subacute catarrh of the stomach rather than upon a simple functional disorder of the digestive organs.

Analysis of the Symptoms.

Pain and Flatulence.—The first symptom which attracts attention is extreme fretfulness and irritability of the infant during the process of digestion. Under normal circumstances, as soon as a meal is finished, the child falls quietly asleep, and remains in a more or less somnolent condition until the next meal is due. But when indigestion occurs, the infant either continues wakeful and restless, or moans and twitches in its sleep. In mild cases, the chief objective signs of abdominal discomfort consist of a series of facial contortions and restless movements of the limbs. In such it may be noticed that the infant suddenly elevates the angles of its mouth, everts the upper lip, raises the eyebrows, and perhaps frowns as though in doubt or anger. These grimaces are often accompanied by short cries or moans, which, although capable of being temporarily appeased by the presentation of some object of special interest, are apt to recur as soon as the infant wearies of the novelty and reverts once more to its bodily discomfort. In more severe cases the child gives vent to a succession of piercing shrieks, which may continue until it is forced to desist from sheer exhaustion. The face is pale and wears an agonized expression, sweat appears upon the forehead and scalp, the knees are drawn up to the abdomen, and the whole body may appear to writhe under the influence of pain. These symptoms persist for a variable length of time, and either subside in a gradual manner before the advent of the next meal, or disappear suddenly after the discharge of a quantity of flatus by the bowel. It was formerly believed that the painful phenomena were caused by the irritation of the stomach and intestine by masses of curdled milk which had escaped digestion. It appears, however, to be much more probable that the products of fermentation rather than the food itself are directly responsible for the dyspeptic symptoms. The irregular distension of the abdomen which usually occurs in these cases after meals, as well as the loud borborygmi, which can often be heard during an attack of pain, indicate that the intestine is excited to spasmodic efforts to rid itself of the accumulated flatus, and the rapid disappearance of the pain when this has been effected demonstrates the colicky nature of the symptom.

---

[1] Loewy, *Wien. Med. Fresse*, 1883, s. 1542.

[2] Ueffelmann, 'Handb. d. Hygiene des Kindes,' 1881, s. 233.

Nausea and Vomiting. — Vomiting after food is extremely common during the period of infancy, and may arise from causes quite unconnected with disease of the digestive organs. Thus, medical advice is frequently sought on account of a form of sickness which occurs regularly after each meal. But notwithstanding the fact that a certain quantity of milk is constantly rejected in this manner, the child continues to thrive and to gain in weight, while the most careful examination of the abdomen and the vomit fails to demonstrate the presence of any abnormal condition of the stomach or its secretion. A rational explanation of this phenomenon is not difficult to find when the habits of the child are made the subject of careful study; for it is usually found that, owing either to natural greediness or to careless indulgence on the part of the nurse, the infant habitually imbibes at each meal an amount of milk far in excess of its gastric capacity, with the result that the stomach becomes over-distended for a time. At a later period of life such a transgression would be followed by acute dyspepsia, but in the infant this danger is averted by the anatomical peculiarities of the organ. The vertical position assumed by the stomach during the first ten months of life, combined with the deficient development of the fundus and of the cardiac sphincter, endows the organ with a kind of automatic regulating power; for as soon as the pressure in its interior reaches a certain point the muscular walls are thrown into reflex contraction, and the surplus milk regurgitates into the mouth. Vomiting immediately after meals also occurs in cases of cerebral inflammation or tumour, the food being pumped up almost as soon as it is swallowed. But in these cases the presence of special cerebral symptoms, and the absence of gastric disease, permits of an accurate diagnosis being made of the cause of the emesis. Under exceptional circumstances the accidental absorption of mineral poisons, or a natural intolerance of certain drugs, is responsible for the existence of the continued vomiting.

In mild cases of dyspepsia vomiting is seldom a constant symptom ; indeed, one may go so far as to say that, whenever emesis constantly follows the ingestion of food, catarrh of the mucous membrane of the stomach is present. In chronic dyspepsia, on the other hand, vomiting is often observed during the first hour which follows the meal, and is usually accompanied by nausea, if the languid, pallid and faint appearance of the child, which usually precedes the act of emesis, can be accepted as an indication of the feeling of sickness. The milk which is rejected on these occasions is considerable in quantity, curdled, and extremely sour-smelling. It is acid in reaction, often frothy in appearance, and contains a large quantity of lactic acid. In many cases the solid residue is retained in the stomach despite continued retching, and the infant merely rejects some clear fluid, the exit of which is accompanied by the evolution of gas. Whenever the vomiting continues after the organ has rid itself of the remains of its last meal, and especially if mucus is present in the ejecta, it may be confidently assumed that the original functional disorder of the stomach has given rise to gastric catarrh.

Diarrhœa. — Except in those rare cases where, owing to repeated vomiting, the disorder remains strictly limited to the stomach, the continued entrance of fermenting material into the small bowel tends to arrest the process of intestinal digestion, and to give rise to appreciable changes in the general character of the stools.

The first change to be observed is the substitution of grayish-white cheesy motions for the bright yellow and homogeneous material of which the evacuations of a healthy infant are usually composed. This arises from deficient digestion of the milk, and the consequent elimination of an excess of casein and fat. At a later period the existence of intestinal fermentation makes itself apparent by the frequent passage of frothy and sour-smelling motions mixed with much fœtid gas. At this stage of the complaint the infant often passes from five to eight stools in the course of the twenty-four hours, and suffers periodically from flatulent distension of the abdomen and attacks of colicky pain. As the disorder becomes established the tendency is for the stools to assume a distinct green colour, and at the same time to become looser in character and less acid in reaction. The exact tint varies considerably, both in different cases and also in the same case from time to time. Thus, in some instances the motion presents a variegated colour from the admixture of green with flakes or spots of yellow, while in others the whole evacuation closely resembles chopped spinach, both in colour and consistence. In rare cases the stool consists of a muddy green liquid. Duodenal indigestion is stated by certain writers to be accompanied by pale yellow stools which have a peculiar glistening appearance, owing to the excess of fat which they contain.[1] This 'fat diarrhœa' is, however, seldom encountered in ordinary cases of fermentative dyspepsia. The green colour of the motions has given rise to considerable discussion. According to Lesage and Hayem,[1] it arises from the presence of a special chromogenic bacillus, which can be isolated from the fæces and cultivated in artificial media. Although this micro-organism may occasionally be the cause of the green colour of the stools, in the vast majority of the cases it is absent from the evacuations, and consequently cannot be considered as even a common cause of the phenomenon.[2] On the other hand, the presence of bile in the green stools can invariably be demonstrated, so that it would appear probable that some change in the colouring matter of the hepatic secretion is responsible for the peculiar coloration of the fæces. In this connection the researches of Pfeiffer[3] are particularly interesting. This observer has shown that in the presence of an alkali bilirubin is readily converted into biliverdin, while the converse can only be brought about through the instrumentality of strong acids. He has also shown that in cases of green diarrhœa the stools are always less acid than normal, and may sometimes even possess an alkaline reaction and ammoniacal odour; and that even when they are mixed with acid contents of the large intestine the degree of acidity they acquire is insufficient for the reconversion of the biliverdin into bilirubin. It would therefore seem probable that in many cases of simple dyspepsia the contents of the small intestine undergo a kind of alkaline fermentation, the products of which convert the yellow colouring matter of the bile into biliverdin. Frequent watery motions, or motions containing an excess of mucus, are more commonly the result of catarrhal affections of the intestine than of simple disturbance of its digestive functions.

---

[1] Demme, *Jahresbericht des Kinderspitales in Berne*, 1877; Biedert, *Jahr. f. Kinderheil.*, xii., s. 197.

---

[1] Lesage, *Revue de Méd.*, xiv., p. 1030; Hayem, *Bullet. Thérap.*, 1887, p. 441.

[2] Jeffries, *Trans. Amer. Pediat. Soc.*, i., 1889, p. 249; Baginsky, *Deut. Med. Woch.*, 1889, s. 391. (I have also examined fourteen cases for the green bacillus with a negative result.)

[3] *Jahr. f. Kinderheil.*, xxviii., s. 164.

Appetite.—As long as the digestive disorder remains uncomplicated by gastric catarrh, the infant continues to take its food with avidity, and may even exhibit an abnormal craving for milk. This is usually held to indicate that the appetite is undiminished, and may even be exaggerated. Judging, however, from the subjective phenomena that accompany acute dyspepsia in adult life, it would appear probable that thirst is a more prominent symptom than hunger, and that it is merely a craving for drink that induces the infant to partake so freely of the breast or the bottle. Indeed, in the acute form of infantile dyspepsia it may usually be observed that cold water is imbibed with equal avidity and with more satisfying results than milk. In most cases the *tongue* remains clean and moist throughout the whole course of the complaint, and it is only in those cases where vomiting or diarrhœa are severe that the organ becomes covered with a creamy fur or shows any tendency to dryness. As a rule, the secretion of *urine* is diminished, and if the motions are frequent and loose, no water may be passed for many hours. Occasionally the urine is retained in the bladder during an access of abdominal pain, and a comparatively large amount is voided either coincidently with or immediately after the expulsion of gas or undigested food from the bowel.

At the commencement of an acute attack of dyspepsia, the *temperature* of the body is often raised a degree or two, but the fever soon subsides, and in chronic cases the temperature is invariably subnormal. *Loss of flesh* occurs in every case, and is especially noticeable and rapid in those which suffer from vomiting and diarrhœa. The neck, buttocks, and thighs are the first parts of the body to show signs of wasting, absorption of the subcutaneous fat in these regions causing the skin to lose its elasticity and to hang in loose folds or pleats. As the disease progresses, the face becomes pinched and pale, the eyes sunken, while the skin round the mouth and over the eyelids acquires a bluish-black tint. The hands and feet are habitually cold, and the skin ceases to perspire, and becomes harsh, dry, and inelastic. The *pulse* is feeble, and often diminished in frequency, especially during the periods of digestion. An abnormal frequency of the heart-beat is usually indicative of severe exhaustion, or of some catarrhal affection of the digestive organs.

Few infants suffer from indigestion without exhibiting some *nervous symptoms* of reflex origin. In the simplest variety the muscles of the extremities are thrown into tonic spasms coincidently with an attack of abdominal pain. The arms are adducted, the forearms flexed, and the fingers firmly clenched over the thumbs. The legs are extended, the soles of the feet inverted, and the head inclined toward one or other shoulder. At the same time the face becomes markedly pale, the mouth twitches slightly, and the eyeballs are rotated upwards, and squint. Symptoms of this nature are extremely common in rickety and debilitated children who are the subjects of painful dyspepsia, and are commonly alluded to by nurses as 'internal convulsions.' They may only occur once or twice during the course of the digestive complaint, or they may ensue after each meal. Nervous phenomena of a more serious nature are apt to accompany the more acute phases of the disease. These consist of one-sided convulsions of an eclamptic type, which recur at short intervals, and are accompanied by periods of insensibility. Their onset is often preceded by severe retching and diarrhœa, and in this respect they closely resemble the epileptic seizures which sometimes affect adults suffering from chronic dilatation of the stomach.[1] Occasionally death occurs during an attack from failure of respiration.

Another symptom of reflex origin is to be observed in the short, dry *cough* which so often follows the ingestion of food in dyspeptic infants. This may continue during the whole period of digestion, but more often subsides abruptly after an attack of emesis or diarrhœa. The subjects of acute indigestion are also liable to a spasmodic form of *dyspnœa*, to which the name of 'asthma dyspepticum' has been given. In this condition the respiration is quick and laboured, the pulse feeble, and the face and extremities often livid. No physical signs are to be found on examination of the chest, but the stomach and large bowel are frequently distended with gas, and the symptoms rapidly subside when this has been expelled or the contents of the stomach evacuated.

---

[1] Author, *Trans. Clin. Soc.*, vol. xxviii., p. 13.

CHEMISTRY OF DIGESTION.

There appears to be some disagreement among different writers concerning the activity of the gastric secretion in cases of infantile dyspepsia. Thus, Clopatt[1] states that in only one case out of the six he examined was he able to detect any diminution in the amount of the hydrochloric acid, or the presence of secondary organic acids. Leo,[2] on the other hand, ascribes the excessive acidity of the gastric contents which he always observed to the presence of the products of fermentation. Among the seven cases examined by Cassel[3] free hydrochloric acid was constantly absent from the gastric contents in two; in three it was occasionally present, while in the remaining two the secretion appeared to be normal. As the result of his researches upon the subject, Van Puteren[4] believes that in the great majority of the cases a noteworthy diminution occurs in the secretion of the hydrochloric acid. My own observations upon this point were conducted upon twenty-two infants varying in age from eleven days to four months. In three instances the disorder had existed for less than five days, while in the remaining nineteen the complaint had reached a chronic stage. In each case the contents of the stomach were evacuated and examined an hour and a half after the administration of the test meal. As a rule, the quantity of material extracted from the stomach exceeded the normal, and filtration was rendered difficult owing to the presence of curd and mucus.

---

[1] Clopatt, *Finska läkeresälisk. handl.*, xxxiv., s. 1.

[2] Leo, *Berl. Klin. Woch.*, 1889, s. 49.

[3] Cassel, *Arch. f. Kinderheil.*, xii., s. 175.

[4] Puteren, *Dissert.*, St. Petersburg, 1889.

In each of the three cases of the acute disorder, free hydrochloric acid was absent from the contents of the organ, while the extracted material was found to contain a large quantity of lactic acid. Out of the nineteen cases of chronic dyspepsia, the mineral acid in a free state was detected in seven, but in each instance the phloroglucine-vanillin reaction was but faintly visible. The total acidity of the mixture was usually below the normal, and as a rule varied between 0·135 and 0·054 per cent. HCl. Upon a few occasions the total acidity was abnormally high, owing to the presence of the acid products of fermentation. Combined hydrochloric acid, as estimated by the process of Hehner and Seeman (see Appendix), never exceeded 0·118 per cent. In every case the presence of lactic acid was demonstrated by the help of Ueffelmann's solution, and in five cases butyric acid was also detected. Pepsine and rennet were invariably present, although deficient in quantity. One of the most noticeable features in these cases was the length of time the food remained in the stomach. It has already been observed that under normal conditions the stomach is usually free from food within one and three-quarter hours after a meal (p. 13); but in cases of chronic dyspepsia it is often possible to extract several cubic centimetres of curdled milk two and a half or even three hours after the last meal. It would therefore appear that in cases of functional derangements of the stomach in infancy the process of gastric digestion presents several striking deviations from the normal, of which the most important are a diminution in the secretion of hydrochloric acid, and the production of organic acids by fermentation of the food. As a result of these changes the food is only partially digested, and is retained in the stomach for an undue length of time.

PROGRESS AND TERMINATION.

Acute dyspepsia, occurring in an otherwise healthy infant, usually runs its course in two to five days, a natural cure being effected by the expulsion of the indigestible or fermenting material from the body by vomiting and diarrhœa. If the warning conveyed by the disease is regarded, and the dietary placed for the future under judicious supervision, the child will rapidly regain its former state of health, and may eventually appear to be none the worse for its illness. It must be noticed, however, that one attack always predisposes to another, so that an infant which has once suffered from dyspepsia may subsequently develope the complaint from some trivial alteration in its diet, or, should the summer be at hand, fall a victim to an attack of acute catarrh of the stomach and intestine.

The more insidious and chronic form of dyspepsia never terminates by natural cure. On the contrary, a persistence in those errors of diet which originally initiated the complaint gradually destroys the functional activity of the digestive organs, and either occasions a fatal form of malnutrition or else paves the way for an attack of some serious intercurrent disease. The former result is most commonly observed in those cases where the digestive disorder has arisen from the wholesale administration of farinaceous foods. In such, vomiting is seldom observed, while the bowels are confined rather than loose in their action ; but the want of assimilable food, combined with the ill effects of the digestive disorder, induces a steady loss of flesh, so that eventually the infant is reduced to a condition of profound malnutrition, and dies either from asthenia, syncope, convulsions, or from some other result of chronic starvation. In other cases, and more especially in those where the functional disorder has been provoked by the injudicious use of milk-foods, catarrhal inflammation of the digestive tract is very apt to be set up by the products of food fermentation ; and the infant is either carried off by an acute attack of intestinal catarrh, or falls a victim to that progressive and incurable form of marasmus which arises from atrophy of the mucous membrane of the stomach and intestine.

COMPLICATIONS.

Among the complications of chronic dyspepsia *gastro-intestinal catarrh* is by far the most important. When this occurs in an *acute* form, the infant vomits after each meal, and even in the absence of food the stomach will often reject considerable quantities of bile-stained mucus. The bowels are loose, and the stools watery and largely mixed with mucus. The temperature of the body is elevated, the pulse quickened, the tongue furred, and signs of exhaustion soon become apparent. When the infant has already been debilitated by chronic dyspepsia, an attack often proves fatal.

Chronic gastro-intestinal catarrh commences in a more insidious manner, and usually persists for many weeks. In its early stages it is susceptible of cure, but as soon as changes of an organic nature have taken place in the mucous membrane of the digestive tract, a fatal form of marasmus usually results.

Bronchial catarrh is another frequent accompaniment of simple dyspepsia. Thus, I find that, among the hundred cases of the disorder already referred to, no fewer than thirty-seven presented the symptoms and physical signs of bronchitis, while in seven others broncho-pneumonia was also present.

Infants suffering from chronic indigestion are also apt to suffer from irritable *skin affections*, such as eczema, urticaria and strophulus. The former is usually encountered behind the ears, upon the scalp, or about the folds of the skin and flexures of the joints. The two latter make their appearance at irregular intervals, and are often observed only at night-time after the body has been washed. Urticaria usually affects the trunk and extremities, while the strophulous eruption is most common upon the thighs, sides of the chest, and back.

Parasitic *stomatitis* is very apt to attack the subjects of dyspepsia. Sometimes thrush is observed to precede the development of the acute disorder, and in such cases it is possible that the introduction of the vegetable parasite into the stomach may have been partly responsible for the origin of the gastric fermentation. In the majority of the cases, however, the disease of the mouth is the result, and not the cause, of the dyspepsia, and is induced by the abnormally acid state of the buccal secretions favouring the development of the fungus.

The malnutrition which results from non-assimilation of the food renders the infant extremely vulnerable to an attack of an *infectious fever*, or, during the hot months of the year, to the epidemic form of intestinal catarrh. Thus, among my hundred cases of dyspepsia, I find that thirteen contracted whooping-cough, nine measles, and three scarlatina, during the few weeks they remained under treatment at the hospital. It must be remembered, however, that the chances of infection are particularly great among children who are crowded together in the waiting-room of a hospital.

Diagnosis.

Since the invasion of almost every specific febrile complaint is apt to be accompanied by vomiting and diarrhœa, it is possible to mistake the initial stage of these diseases for simple indigestion. In such cases, however, the general symptoms are much more severe than those which accompany the functional disorder of the digestive organs, the temperature being high, the pulse quick, the tongue furred, while within a short time the special phenomena of the complaint manifest themselves.

A differential diagnosis between acute dyspepsia and acute gastric catarrh is a matter of greater difficulty, and, indeed, there is every reason to suppose that many cases diagnosed as ‘simple indigestion’ are in reality instances of mild gastric catarrh. In the more severe varieties of the inflammatory disease a correct diagnosis can easily be arrived at by observance of the persistent temperature, the incessant retching and vomiting of mucus, the frequent and watery character of the diarrhœa, and the early development of the symptoms of collapse.

Chronic dyspepsia has to be distinguished chiefly from chronic gastro-intestinal catarrh. In this latter disease vomiting is a common symptom, and the material rejected from the stomach contains an excess of mucus. Diarrhœa is invariably present, and the stools are often passed with much pain and straining. The tongue is furred, the appetite deficient, and the temperature of the body usually elevated towards evening. The soft tissues waste rapidly; and as the infant becomes exhausted it is apt to exhibit convulsions and other nervous symptoms. Even under favourable conditions recovery is usually slow and imperfect, while in the majority of the severe cases death results from progressive asthenia. In simple dyspepsia, on the other hand, abdominal pain and flatulence constitute the chief symptoms of the complaint. The temperature is usually depressed, the pulse slow, while vomiting and watery diarrhœa are seldom prominent symptoms of the case. Recovery also is rapid and complete as soon as the diet has been suitably adjusted.

TREATMENT.

General.—Symptoms of indigestion occurring in an infant always demand immediate attention, and no case is too trivial to be ignored. The infant should be confined to its cot as much as possible, and all tossing about in the arms, which obtains so much favour among nurses as a means of soothing irritable children, should be prohibited, since shaking of the body favours the formation of large curds in the stomach, and thus excites abdominal pain and vomiting. Owing to the increased susceptibility shown by dyspeptic children to the influence of cold, the air of the nursery should be maintained at an equable temperature (70° F.). A warm bath may be given each night and morning, but care must be taken on each occasion to dry the skin rapidly and thoroughly with a warm towel. During the attacks of abdominal pain a hot fomentation or a linseed poultice may be applied to the abdomen ; and in severe cases a few drops of laudanum or turpentine, or a little mustard, may be sprinkled over the surface of each application. In cases of moderate severity gentle friction with the hand in the direction of the colon helps the bowel to expel its gaseous contents, and thus tends to relieve the pain. In feeble infants an attack of colic sometimes induces the symptoms of collapse. When this occurs, it is usually best to place the child at once in a hot bath containing mustard, and afterwards to apply friction to the skin by means of a warm salted towel, or to envelop it in a warm blanket, with hot-water bottles at the sides and feet.

In every case the state of the mouth should be made the subject of special attention. After each meal the buccal cavity should be carefully cleansed from any food retained between the lips and gums, by means of a piece of soft linen wet with warm water. If signs of thrush make their appearance, glycerine of borax may be used, or some dilute antiseptic solution, such as resorcine (ten grains per ounce), hyposulphite of sodium (sixty grains per ounce), sulphurous acid (1 in 6), or salicylic acid (1 in 250), may be employed. Even when the immediate symptoms of dyspepsia have quite subsided, special care must be taken to protect the infant from exposure to cold. With this object, the dress should be made to completely cover the arms and neck, while stockings should be provided instead of the ordinary short socks. Such elementary precautions are usually regarded with disfavour by those persons who are unable to appreciate the difference between care and coddling, and who imagine that children can be 'hardened against disease' by allowing them to parade about in the open air clothed in the scantiest and thinnest of garments. It is owing to this, and other barbaric superstitions concerning the management of infants, that cases of simple indigestion are so often fostered, and eventually converted into serious, and even fatal, cases of gastro-intestinal catarrh.

Dietetic.—Notwithstanding the fact that dyspepsia is almost invariably the result of some error either in the preparation, mode of administration, or quality of the food, a radical change of diet should never be adopted as a routine practice. This caution is particularly necessary in the case of *infants at the breast*, among whom indigestion commonly arises from causes which can be readily adjusted by the expenditure of a little care and trouble. The habit which prevails among many practitioners, of insisting upon an infant being weaned as soon as it shows any signs of indigestion, is one which cannot be too strongly deprecated, since, as a rule, it is not only an unnecessary procedure, but one which entails the administration of some artificial form of food which in itself is far less digestible than the original milk. It consequently happens that in the majority of such cases the last state of the infant is worse than the first.

In *acute* cases the state of the mother's health and the condition of her milk require to be investigated, and should it be found, as is often the case, that the symptoms of the infant are dependent upon some temporary change in the quality of the milk (p. 23), the child need only be deprived of the breast for a short time, and in the meanwhile be supplied with some suitable substitute for its mother's milk. Where the digestive disorder can be traced to over-suckling, it is only necessary to regulate the frequency and quantity of the meals in order to insure a rapid cure of the complaint. If, on the other hand, the mother is found to be suffering from some chronic disease which will probably cause a *permanent* deterioration of the mammary secretion (p. 24), or if after repeated trials the milk still continues to disagree, it is wise to procure an efficient wet-nurse at once, or, should this be impossible, to substitute the bottle for the breast. The same rules hold good with respect to infants suckled by wet-nurses (p. 33).

Dyspepsia occurring in *hand-fed children* may also be due to some trivial and remediable cause, so that if the food itself appears to be suitable, it may again be tried after the symptoms of indigestion have subsided. Should they recur, however, the diet must be changed.

In cases of *severe or obstinate dyspepsia*, it is usually necessary to afford the digestive organs a period of physiological rest by withholding food entirely for about twenty-four or thirty-six hours. During this time the infant may be allowed small quantities of barley or rice water (R. 1), albumin-water (R. 4), or thin gruel from a spoon, to which, if symptoms of exhaustion are present, a little diffusible stimulant may be added. As a rule, these liquids should be given *cold* at first, being only warmed to the temperature of the body when marked improvement has set in. The commencement of recovery is often indicated by the infant objecting to partake any longer of cold food. There is a strong superstition among nurses that a child cannot be deprived of milk for more than a few hours without serious consequences, so that, unless the prohibition of food is made most emphatic, they will often encourage the progress of the complaint by the surreptitious administration of milk-gruel or some other equally deleterious compound.

As soon as the immediate symptoms have abated, a cautious trial may be made of some easily digestible form of nourishment, and the child may be allowed to suck the breast for a few minutes, or to take small doses of whey, sterilized milk diluted with three times its volume of barley-water, or equal parts of 'strippings' and water. In other cases small quantities of veal, chicken, or mutton broth may be allowed (R. 8).

If the infant is able to digest these without difficulty, the food may be gradually increased in strength by the addition of cream to the whey or milk, or by a diminution in the proportion of barley-water, until eventually the child is able to take the full diet suitable to its age and requirements (p. 43).

The caution which characterizes these procedures is absolutely necessary on account of the enfeebled condition of the digestive organs which persists for some time after the subsidence of the prominent symptoms of dyspepsia. Any attempt to force the child to take more than it can digest is usually followed by a recurrence of the dyspeptic symptoms or by the development of acute catarrh of the digestive tract.

Medicinal.—There are three principal indications for the medicinal treatment of simple dyspepsia in infants. The first is to promote the elimination of the irritant contents of the stomach and intestine; the second, to allay the pain and other prominent symptoms of the disorder; and the third, to stimulate the powers of digestion which have been enfeebled during the course of the complaint.

All cases of acute dyspepsia exhibit a natural tendency toward self-cure through the medium of the vomiting and diarrhœa, which serve to evacuate the fermenting contents of the stomach and bowel. The first duty of a medical man is to ascertain if Nature has completed its task, and, if it has not, to afford it the necessary aid.

If the stomach is empty, attention should be concentrated upon the state of the intestine; but if vomiting has not occurred, and the child exhibits a sour-smelling breath, a coated tongue, fulness of the epigastrium, or other signs indicative of gastric fermentation, it is advisable to administer an emetic, or to wash out the organ with warm water. This latter measure is chiefly indicated in the more chronic forms of the complaint, where dilatation of the stomach is present. It may therefore be stated as a general rule that emetics are most useful in the acute, and lavage in the chronic, forms of dyspepsia.

The ease with which emesis can be provoked varies in different cases, but, as a rule, a full dose of ipecacuanha (one drachm every ten minutes), sulphate of zinc (ten grains) or sulphate of copper (half a grain every five minutes) answers the purpose well. Apomorphine is too uncertain in its action when given in small doses, and too depressant when given in large doses, to be employed in these cases. In every instance it is necessary to procure a thorough evacuation of the bowels, especially if the continued existence of intestinal irritation is shown by the presence of colicky pain or the frequent passage of unhealthy stools. For this purpose some laxative like castor-oil (one drachm), the sulphate and carbonate of magnesia (F. 1), rhubarb and soda (five to ten grains), calomel (two grains), or the compound liquorice powder (thirty to sixty grains), may be given, and the dose repeated if the symptoms continue. In some cases, after the initial purgation, the administration of frequent small doses of these laxatives is of greater value than a repetition of the full dose. It rarely happens that these measures, combined with strict regulation of the diet, fail to effect a cure.

Relief of Symptoms

Pain.—In cases of a subacute or chronic nature the child may continue to suffer from attacks of colicky pain in the abdomen for some time after the subsidence of the more urgent symptoms. This arises from the persistence of fermentation in the intestine. The readiest means of affording relief is by the administration of ten to fifteen drops of spirits of nitrous ether, or drachm doses of chloroform, peppermint, or dill-water. When symptoms of collapse accompany the attacks of abdominal pain, the infant should be at once put into a hot mustard bath, and friction applied to its extremities, while at the same time ten to fifteen drops of brandy or sal volatile may be given in warm water. As soon as reaction has set in, the child should be removed to bed, enveloped in a blanket, and a hot poultice or mustard-leaf applied to the abdomen. Sedatives are rarely required except in obstinate cases, where it is sometimes advantageous to inject half an ounce of warm water containing one or two drops of laudanum into the rectum.

Vomiting.—This symptom rarely continues after the diet has been suitably adjusted. Occasionally, however, an infant who has suffered from acute dyspepsia will habitually reject a portion of its food after each meal, or will vomit any article which is distasteful to it. Some authorities recommend the administration of bismuth, alkalies, or sedative drugs to correct this, while others advise the employment of the gastric douche or of lavage. For my own part, I am in the habit of regarding the phenomenon as a natural defence against a recurrence of the gastric disorder, and one which indicates the necessity for greater circumspection in the choice of the dietary rather than for medicinal treatment. Persistent and severe vomiting, on the other hand, suggests the existence of gastric catarrh, and must be treated in the ordinary way.

Diarrhœa.—As a rule, the intestinal evacuations decrease in frequency as soon as the irritant contents of the bowel have been removed by the purgative remedies. But it sometimes happens that, in spite of repeated doses of castor-oil, the stools still continue to be frequent, liquid and offensive. In such cases recourse should be had to calomel, which may be given in doses of one-eighth to one-sixth of a grain every three hours, either alone or combined with two or three grains of subnitrate of bismuth. In these doses calomel appears to exert an antiseptic influence in the intestine, possibly by its conversion into the perchloride and sulphide of mercury, and in most cases its administration is followed by rapid improvement in the condition of the stools. Should it fail to check the diarrhœa, resorcine (three to five grains every four hours) will usually succeed. Naphthalin is less pleasant to the taste, and not so reliable as resorcine; but occasionally salicylate of bismuth (three to five grains) may be given with advantage. Formerly astringent remedies were employed in all cases of diarrhœa, but they have fallen into disuse since our knowledge of the etiology of intestinal dyspepsia has become more exact. Opium is chiefly indicated in cases where the stools are very numerous and watery, and are accompanied by symptoms of exhaustion. In such cases compound powder of ipecacuanha, combined with chalk and bismuth (F. 2), may be given at short intervals, or the tincture of opium or nepenthe (half to one drop) may be employed.

Convulsions. — These seldom require any special treatment, since they subside spontaneously as soon as their exciting cause has been removed. Should they prove unduly severe, a few minims of oil of turpentine may be added to each dose of the castor-oil mixture, or chloral and bromide of potassium may be given in the form of an enema (F. 4).

Constipation.—In the case of infants at the breast, the tendency to constipation is easily corrected by the occasional administration of a teaspoonful of manna or Mellin's food dissolved in milk; or a little fresh cream, oatmeal-water, or a dose of salad-oil may be given from time to time. The mother should also be encouraged to drink a tumblerful of 'strippings' occasionally before nursing, or to take porridge at her breakfast. In hand-fed children ten grains of phosphate of sodium or five grains of magnesia may be added to the bottle once or twice a day, and the abdomen rubbed each evening with a liniment of soap and aloes (F. 5). A small piece of soap inserted into the rectum each morning often induces a normal motion within a short time. In more obstinate cases active remedial treatment should be commenced by a brisk purge with calomel, castor-oil, or rhubarb and soda. When the rectum is blocked with hard masses of fæces, an enema usually answers best (soap-and-water, four ounces), to which may be added, if necessary, one or two drachms of olive or castor oil. The daily use of enemata, however, must be avoided, as they tend to produce atony of the colon, and so to increase the habit of constipation. In order to procure a daily evacuation, recourse may be had to the compound magnesia mixture (F. 1), the compound powder of liquorice (one drachm), the compound decoction of aloes, syrup of senna, the confection of senna and sulphur, or to the sulphur lozenges. If the motions are abnormally pale, an occasional dose of calomel or gray powder may be given, or a few drops of the tincture of podophyllin be administered after meals. Of all laxatives, cascara sagrada is the most valuable, and can be advantageously given in the form of the liquid extract (five to fifteen minims), combined with a teaspoonful of maltine, twice a day after meals. As soon as the bowels act each day, the purgative remedy should be gradually diminished, and a tonic, such as nux vomica, perchloride or sulphate of iron, dialysed iron, or calumba, be substituted for it. In some cases the administration of pepsin after the meals appears to assist the intestinal action by aiding the process of digestion.

After-Treatment.

As soon as the tongue is clean, and the stools present a normal appearance, tonics should be exhibited with the view of improving the general health and the condition of the digestive organs. For this purpose the tartrate and citrate of iron are invaluable. Arsenic answers well in many cases, while in others a small dose of ipecacuanha wine, combined with nux vomica and ginger, after each meal, proves an excellent stimulant to digestion (F. 6). Quinine usually disagrees. Cod-liver-oil is an extremely valuable remedy, especially when combined with iron, but it must not be commenced until the diarrhœa has completely subsided. In all cases it is wise to omit the tonic treatment once a week, and to administer a small dose of gray powder at night, or a mixture containing rhubarb and taraxacum (F. 7).

Pepsin is chiefly indicated when digestion continues slow and difficult, and is accompanied by flatulent distension of the abdomen toward evening. The best preparation is the acid glycerine of pepsin (Bullock), but occasionally the dry powder or lactopeptine answers well. The liquor pepticus and the liquor pancreaticus (Benger) are sometimes of use, but as a rule they are inferior to the aforementioned. Dilute hydrochloric acid was strongly recommended by Henoch for the after-treatment of simple diarrhœa; it may be given in doses of five to ten minims of the dilute preparation, combined with syrup and a carminative, after the meals. It is contra-indicated in cases of catarrh of the stomach and intestine.

CHAPTER IV.

ACUTE GASTRO-INTESTINAL CATARRH IN INFANTS

(INFLAMMATORY DIARRHŒA—ZYMOTIC DIARRHŒA—CHOLERA INFANTUM).

DURING the first two years of their life, children are very prone to suffer from inflammation of the alimentary tract, which affects, with a varying degree of intensity, the whole of the inner surface of the digestive canal from the cardiac orifice of the stomach to the anus. Since at this period of life, however, the stomach is only partially developed, and is as yet far removed from attaining that important place in the systemic economy that it occupies at a later date, the symptoms of the complaint are intestinal rather than gastric, and in all cases the disordered condition of the bowel constitutes the most prominent feature of the disease. The pernicious habit of designating a disease according to its principal symptom has led to the inclusion of a large number of distinct complaints accompanied by a loose state of the bowels under the term of 'diarrhœa,' the significance of which is still further confused by such additional qualifications as 'simple,' 'inflammatory,' 'zymotic,' etc. In order to avoid the employment of a term which is only productive of error, I propose to consider the various acute diseases of the digestive tract associated with catarrhal inflammation of its mucous membrane under the single term of 'acute gastro-intestinal catarrh,' pointing out, as far as our present knowledge will permit, the pathological and clinical distinctions which exist between the different members of the series.

ETIOLOGY.

Acute gastro-intestinal catarrh in infancy arises from two principal causes : (1) irritation of the mucous membrane of the stomach and intestine by undigested and fermenting food ; (2) absorption into the general circulation of certain organic poisons produced by microbic activity. In the former case the severity of the symptomatic disease is directly proportionate to the intensity of the local inflammation, but in the latter no constant relation can be demonstrated between the degree of inflammation and the intensity of the general symptoms. For the purposes of description, cases of acute gastro-intestinal catarrh may be divided into two classes—'simple' and 'infective'—according as the complaint appears to arise from local irritation of the digestive tract or from a general toxæmia.

The Simple Variety. — Acute gastro-intestinal catarrh arising from fermentation of the food is almost entirely confined to hand-fed children, being seldom observed in those nourished exclusively by the breast. Sucklings are, however, extremely susceptible to the complaint at the period of weaning, since at that time they are suddenly exposed to all the dangers of food contamination without having acquired that degree of tolerance which the use of artificial foods confers after a time upon hand-fed infants. With regard to the nature of the food which is most prone to excite an attack, there is little to be added to what has already been stated concerning the etiology of simple dyspepsia. Indeed, the whole subject may be summed up in the general statement that the administration to an infant of any form of food which is difficult of complete digestion and absorption is liable to excite fermentation, and thus to produce catarrh of the alimentary canal. This eventuality is greatly favoured by bacterial infection of the food during the process of its manufacture or administration.

Formerly it was held that the catarrhal disease which results from defective feeding was due to the mechanical irritation of the mucous membrane by masses of casein and other particles of food which had escaped solution by the digestive juices. It is now, however, generally accepted that the agency responsible for the complaint is a chemical rather than a mechanical one, and that the diminished secretion of hydrochloric acid which accompanies most functional disorders of the stomach permits fermentation of the food to proceed unchecked to the formation of chemical substances of a deleterious nature. Among the latter, lactic, acetic, and butyric acids are known to be capable of inducing severe inflammation of the mucous membrane of the alimentary tract when brought into contact with it in a sufficient degree of concentration, and it is also probable that many bye-products of proteid decomposition are endowed with similar properties. The catarrhal disease is both predisposed to and aggravated by the liberation of gas during the process of fermentation, which distends the stomach and intestines and prevents the expulsion of their noxious contents by peristaltic contraction. It should also be remembered that the failure of nutrition that results from chronic indigestion is in itself a powerful predisposing cause to an attack of acute catarrh.

The infantile habit of inserting every tangible article into the mouth is occasionally responsible for the development of symptoms closely resembling those of acute catarrh of the stomach and intestine. Thus, cases have been recorded where the swallowing of peas, beads, pebbles, pieces of eggshell, chalk, and even hair, was followed by vomiting and purging, which persisted until the offending substances had been expelled; while in not a few instances the sucking of painted toys, or the ingestion of mineral poisons in other ways, has given rise to severe, and even fatal, inflammation of the alimentary tract.

The Infective Form.—Most of the specific infectious diseases of childhood are accompanied by catarrh of the stomach and intestine, especially scarlatina, measles, small-pox, typhoid and influenza. In many cases the symptoms of deranged digestion are chiefly in evidence at the commencement of the attack or about the period of crisis, being masked during the height of the disease by phenomena of a more important nature, but occasionally the intestinal complication remains conspicuous during the whole course of the complaint. Exposure to cold is usually stated to be an important cause of acute gastro-intestinal catarrh, and there can be no doubt that an inflammatory affection of the nasal, pharyngeal or laryngeal mucous membrane is often associated with an analogous condition of the stomach and bowel. Cases of this kind are, however, only examples of an infectious form of catarrh, and cannot be assigned to simple chilling of the surface of the body.

Of all the infective varieties of gastro-intestinal catarrh, that which occurs in an epidemic form during the summer months is by far the most interesting and important. The exact etiology of this disease is still shrouded in mystery, since up to the present all attempts to isolate the specific microbe have ended in failure; but the mass of evidence in support of the theory of its specific origin is so overwhelming, that even in the absence of direct confirmatory evidence the infectious nature of the disease may be accepted as a fact.

Among the several conditions which predispose to an attack, an antecedent disorder of the digestive system ranks first. This is shown by the inordinate frequency with which the disease attacks children who are already suffering from some functional or inflammatory form of dyspepsia. Chronic malnutrition is another important predisposing cause to the inception of the infectious disorder, and consequently those infants whose vitality has been lowered by injudicious feeding, syphilis, rickets, or tuberculosis, are usually found among the earliest victims of an epidemic. In like manner, an attack of some specific fever, such as measles, scarlatina, or pertussis, materially increases the natural susceptibility to the complaint. Although the disorder may attack children of any age, it is most common between the ages of six and sixteen months. Among 772 cases observed by Emmett Holt,[1] no fewer than 630 occurred in infants less than two years old.

---

[1] Keating's 'Cyclopædia of Diseases of Children,' vol. iii., p. 61.

The disease may be said to be almost entirely confined to hand-fed children, since of those which derive their entire nourishment from the breast not more than 3 per cent. usually suffer. The inordinate frequency of the complaint in early life can only be explained by the supposition that the dietetic conditions which prevail at this period are eminently favourable to systemic infection through the digestive tract. This is supported by the fact that as soon as some form of artificial food is added to the dietary of a suckling its comparative immunity from the disease at once disappears; while the period of weaning is fraught with special danger to the infant unless the food which is administered has been rendered completely aseptic. Lastly, it may be noticed that the comparative infrequency of the malady after the age of two years coincides in point of time with an increased functional activity of the stomach, and the introduction of a diet which is free from most of the dangers which beset the artificial feeding of infants.

The defective hygienic surroundings of the poor render them particularly prone to fall victims to the epidemic malady, and the mortality is consequently much greater among the dwellers in overcrowded, ill-ventilated, and poverty-stricken households than among those who live under healthier conditions. It is also a common observation that children who reside in the basements and cellars of houses, especially if these are situated in low-lying districts near a river, are more liable to contract the disease than those who tenant the upper stories, or dwell in houses built on higher ground.[1] It is probably owing to one or other of these causes that the epidemic disease is so much more common in large cities than in small towns, and in the latter than in country districts; and while it breaks out at the same season of each successive year in some densely-populated city, the neighbouring villages habitually escape a visitation of the complaint. Numerous attempts have been made to discover a satisfactory explanation of this tendency to periodic recurrence. Careful observations have been made to determine the possible influence of such atmospheric conditions as the degree of humidity, the rainfall, the direction of the wind, and the barometric pressure, upon the incidence of the disease;[2] but the only result has been to show that the study of meteorology affords no solution of the mystery. One fact, however, has been established beyond all doubt, namely, that both the occurrence of an epidemic and the severity of the disease bear a constant and intimate relation to the temperature of the air.

A study of the bills of mortality from 'summer diarrhœa' published in the great cities of Europe and America proves that the death-rate from this complaint undergoes a sudden increase in the month of May, and is gradually augmented until it attains a maximum in July. A slight, though appreciable, diminution occurs in the month of August, and a still further decline in September, so that by the latter end of October the rate of mortality from the disease has regained its normal point. It has also been shown that for the full development of an epidemic the temperature of the atmosphere in the shade must register at least 60° F. for two or three days, and that with every degree above this point the more severe and widely distributed is the resultant disorder. Thus, Vacher[1] has proved that upon every occasion during the course of twenty-five years that the mortality from 'summer diarrhœa' in London exceeded 3 per 1,000, the average summer temperature was greater than 60° F. Another point of considerable interest in this connection is the fact that an epidemic does not develop coincidently with the rise of temperature, but appears to require three or four days of hot weather before its abrupt outbreak. This period of latency has been explained by Ballard[2] upon the hypothesis that the temperature of the soil requires to be heated for a depth of four feet to a temperature of 56° F. before the disease can manifest itself in an epidemic form. It is, however, far more probable that the specific micro-organism is always present in the atmosphere in a comparatively innocuous state, and requires three or four days' incubation at a temperature of 60° F. in order to attain the degree of virulence necessary for the development of an epidemic.

---

[1] Meinert, *Deut. Med. Woch.*, June 14, 1888.

[2] Ballard, *Lancet*, May 4, 1889; Siebert, *Medical Record*, March 24, 1888; Miller, *ibid.*, July 21, 1888; Baginsky, 'Die Verdauungskrankheiten der Kinder,' 1884; Meinert, *op. cit.*

It has been the cause of some dispute why the month of August, which is one of the warmest periods of the year in England, should be accompanied by a lesser mortality than July, and why an epidemic should continue to rage for several days after a fall of temperature has occurred. An answer to the first question is afforded by an appeal to the law which ordains the survival of the fittest; for it is obvious that the weakest members of the community, and those who possess an exaggerated susceptibility to the complaint, will be attacked at the commencement of an epidemic, and their numbers swell the bills of mortality, while the more hardy children will either succumb at a later date or escape altogether. It follows, therefore, that by the time the month of August is reached the ranks have undergone a process of weeding, and the death-rate consequently declines. It is also probable that with the progress of the disease the specific organism itself tends to lose its initial virulence in obedience to some natural law. With regard to the second point of contention, it must be remembered that the seeds of the malady, having been sown broadcast in an extremely active state, cannot be rendered inert immediately the temperature of the air diminishes, but that many deaths must of necessity occur among children who were already suffering from the disease at the time of the change in the atmospheric conditions.

---

[1] Cited by Holt, *op. cit.*

[2] *Op. cit.*

Mode of Infection.—Some writers have attributed the origin of an epidemic to the presence of foul exhalations from the soil, or to an escape of sewer-gas; but although it cannot be denied that the complaint may occasionally be disseminated in one or other of these ways, neither of them can be considered as a constant factor, since, as Ballard has shown, epidemics are equally apt to occur in well-drained towns and in those which possess deficient sanitary arrangements, while the disease is not unduly prevalent in houses where sewer-gas is constantly present. Other authorities have endeavoured to trace the origin of the complaint to contamination of the water-supply, and have even described some special forms of bacteria which were found in the water supplied to the infected households. This theory, however, does not account for the extreme prevalence of the disease among the infantile members of a family, nor for those cases where infants at the breast are attacked while the mother escapes. On the other hand, most of the accumulated evidence goes to prove that the exciting agent of the disease is present in the atmosphere, and gains entrance into the body through the medium of the food or the inspired air. Thus, the introduction of a case into a house or a ward of a hospital is often followed by an outbreak of the malady among the other youthful inhabitants,[1] or the exposure of the stools derived from an isolated case, or of the napkins soiled with the alvine discharges, proves the immediate precursor of an epidemic among the members of a household.[2] Finally, it may be mentioned that the improved methods of feeding in infancy, which aim at the administration of sterile food, have proved the greatest safeguard we possess against the incidence of the disease.

MORBID ANATOMY.

The naked-eye appearances of inflammation are, as a rule, extremely equivocal, and it is often surprising how insignificant are the signs of disease even in cases which presented the most acute and violent symptoms during life. This want of relation between the post-mortem evidence of disease and the severity of the clinical phenomena is one of the most constant and striking peculiarities of inflammatory affections of the digestive tract, and one which can only be explained by the fact that in most instances the mucous membrane undergoes organic changes consequent upon the cessation of the circulation, which have the effect of obliterating the signs of any previous inflammation.

---

[1] Lesage, *Revue de Médecine*, 1887, p. 1030.

[2] Holt, *op. cit.*

As a rule, the stomach is found to be empty after death, or at most to contain a small quantity of a bilious, gruel-like material. In warm weather, when decomposition sets in rapidly, the organ is often found distended with gas. The inner surface is usually coated with a thick layer of mucus, in which a few small blood-clots may perhaps be discerned. When this is removed, the mucous membrane appears swollen and opaque, and in the region of the fundus not infrequently shows signs of post-mortem solution. Sometimes the summits of the rugæ are intensely injected, and present a marked contrast to the pallor of the sulci; in other instances the superficial bloodvessels are distended with blood, and produce an arborescent effect upon the white background. In rare cases the whole of the inner surface of the stomach is swollen and vivid red in colour, with numerous punctiform hæmorrhages scattered through its substance. The small intestine usually contains a certain amount of gas, which by distending its coils partially obscures the larger bowel. Occasionally, however, the transverse colon is also blown out, and protrudes through the wound immediately the abdomen is opened. The contents of the intestine consist of a thin, watery fluid or of a green, odourless slime. The degree of vascular injection varies in different cases, but is always more apparent soon after death than at a later period. In the early stages of the disease the engorgement of the villi gives the inner surface of the gut a bright red colour and a peculiar velvety appearance; but at a later stage the congestion is limited to irregular areas of the mucous membrane, or merely crowns the summits of the valvulæ conniventes, while the intervening portions present a smooth and shining appearance. The solitary glands situated between the secretory structures are swollen, and project upon the surface in the form of small hemispherical masses, while Peyer's patches are likewise enlarged and hyperæmic. Follicular ulceration of the bowel is occasionally encountered in the lower portion of the ileum just above the valve, or in the descending colon and rectum. At first the swelling and softening of the centres of the glands gives rise to a series of small pits in the mucous membrane, each of which is surrounded by a ring of dilated capillary vessels. At a later period the substance of the gland either becomes diffluent, and is discharged into the bowel, or it sloughs *en masse*, and is finally detached, leaving a small circular ulcer measuring from one-sixth to one-third of an inch in diameter. These little ulcers are often so numerous and closely set that the thin bridges of tissue which originally separate one from another break down, and permit the neighbouring sores to coalesce and form irregular patches of considerable size. Occasionally the muscular, or even the peritoneal, coat of the intestine is laid bare during the course of the disease; but as a rule the ulcers maintain throughout their superficial character, and do not penetrate deeper than the submucous tissue. This variety of follicular ulceration seldom occurs until the intestinal catarrh has existed for more than a week or ten days, and its development often adds to the gravity of the case by causing the superaddition of dysenteric symptoms to those of the original disorder.

Gelatinous softening of the stomach and intestines is another frequent result of acute gastro-intestinal catarrh in infancy. Bednar[1] observed softening of the stomach or bowel 100 times in 389 necropsies performed upon infants less than three months of age, and states that of this number no fewer than 61 had suffered from diarrhœa during life. The morbid change is more commonly encountered in the stomach than in the intestine, and in the small rather than the larger bowel. Occasionally the whole of the alimentary tract is affected in a similar manner.

In slight cases the summits of the rugæ in the fundus of the stomach present a somewhat swollen and opaque appearance, and are so soft that the mucous membrane can easily be detached. At a more advanced stage the whole of the mucous membrane in the cardiac extremity of the organ appears to be converted into a grayish or brown mucilaginous substance, which can be wiped off from the subjacent layers with the finger. The superficial bloodvessels are usually distended with blood, and ramify over the surface in the form of black lines. In its most typical form the morbid process affects the entire wall of the stomach either in the form of streaks which run parallel with the rugæ, or as irregular patches situated in the cardiac or middle zones of the organ. In this condition the various coats of the viscus are swollen and transparent, and so friable that the gentlest manipulation is sufficient to tear them. The colour of the affected parts varies in different cases, being sometimes dead white, while in others both the diseased portions and also the healthy mucous membrane present a rose-pink or purplish tinge. Occasionally spontaneous rupture of the stomach occurs during the process of gelatinous softening, and at the necropsy some of the gastric contents can be recognised in the peritoneal cavity, and may even have produced superficial erosion of the spleen or other neighbouring organs. Similar appearances are met with in the intestine, though as a rule they are less obvious than in the stomach, and more often limited to certain coils of the gut than diffused throughout the entire length of the bowel. The affected parts usually present a peculiar anæmic and sodden appearance, while the mucous membrane seems to have been converted into a slimy or glue-like material, which is distributed unevenly over the inner surface of the bowel. Sometimes the wall of the gut is so soft that the tissue falls to pieces during the process of removal from the body. Spontaneous rupture, however, is of extremely rare occurrence.

---

[1] 'Die Krankheiten der Neugebornen,' Wien, 1850, s. 76.

The exact cause of the phenomenon of gelatinous softening has provoked considerable discussion among pathologists, but at the present day it is generally accepted that it arises from autodigestion of the tissues after death. The frequency with which it is encountered in children who have succumbed to disease of the nervous system is supposed to indicate that the hydrochloric acid and peptic ferment are mainly responsible for the post-mortem solution of the tissues. But in cases of acute gastro-intestinal catarrh the secretion of the mineral acid is usually diminished, so that the presence of this factor, at any rate, cannot be regarded as absolutely essential to the process. It would appear from the experiments of Elsässer[1] that pepsin, in combination with acetic acid, is capable of producing even more rapid and extensive softening of the tissues than ordinary gastric juice; and since it is probable that acetic acid is frequently formed in catarrhal diseases of the digestive organs,[2] an easy explanation is thus afforded of the occurrence of the phenomenon. In the *infective* form of the disease the morbid appearances vary considerably. The most common condition is encountered in those cases which succumb during an epidemic of so-called summer diarrhœa, and comprises the ordinary signs of acute catarrh of the stomach and bowel. When, however, death occurs rapidly from collapse (cholera infantum), the macroscopic signs of disease are either ill defined or else entirely wanting. In such cases the small intestine and the transverse portion of the colon are usually distended with gas, while the other parts of the bowel are collapsed or contracted. Any morbid phenomena that may be present are chiefly confined to the jejunum and upper part of the ileum, which are found to contain a moderate quantity of a colourless and almost inodorous fluid closely resembling thin gruel in appearance. The wall of the gut presents a dead-white and sodden appearance, and it requires the aid of a lens to distinguish signs of vascular injection along the summits of the valvulæ conniventes. Peyer's patches and the solitary lymphatic glands of the mucous membrane are swollen and more translucent than normal, while occasionally the mesenteric glands are also enlarged and congested. It is only when life has been prolonged for a day or two that the colon and sigmoid flexure show signs of inflammatory mischief in the form of small bran-like particles of inflammatory exudation upon their mucous surfaces, discrete hæmorrhages, or minute patches of follicular ulceration.

---

[1] 'Die Magenerweichung der Säuglinge,' Stuttgart, 1846.

[2] Baginsky, *Deut. Med. Woch.*, 1888, s. 392.

MORBID HISTOLOGY.

In order to arrive at an accurate conclusion concerning the pathology of any disease, it is necessary to examine not only the organ or organs which appear to be primarily at fault, but also every other important structure in the body. The habit of confining the attention to the morbid appearances presented by any single tissue cannot be too strongly condemned, since it is not only productive of a narrow conception of the pathology of the disease, but encourages the absurd practice of designating a complaint by the title of its most prominent symptom. During the last few years I have been enabled to examine with the microscope more than seventy cases of disease of the digestive organs in children, and in every instance where the intestine was found to be affected with inflammation, indications of a similar condition were encountered in the stomach, whether gastric symptoms were present during life or not.

The Stomach.—The chief signs of catarrhal inflammation of the stomach consist of an engorgement of the superficial vessels and the presence of a round-cell exudation in the interstitial tissue of the organ. The greater portion of the superficial epithelium is detached at an early stage of the complaint, and its columnar cells can easily be recognised in the tenacious layer of mucus which covers the inner surface of the organ. Here and there a few cells still retain their connection with the basement membrane, but these are shrunken and empty or distended with mucus. The fine capillary plexus which surrounds the mouths of the ducts, as well as the vessels situated between the glands, are engorged with blood, and not infrequently small hæmorrhages may be discerned in the superficial parts of the mucous membrane. The lumina of the tubules are blocked with red corpuscles and epithelial cells, and the mouths of the glands are widely dilated and filled with mucus and débris. The outlines of the gastric glands are usually obscured by the dilatation of the bloodvessels and the accumulation of inflammatory products in the connective tissue, but the fundi can be studied without difficulty. As a rule, both the central and parietal cells remain unaffected, and their nuclei stain deeply with hæmatoxylin; but occasionally the peptic cells become detached, and undergo granular or fatty degeneration. The main feature of the section is the extensive infiltration of small round cells into the interstitial connective tissue. This phenomenon does not observe a uniform distribution, but occurs in an irregular or patchy manner near the free surface of the mucous membrane, and extends thence between the tubular glands in the direction of the muscularis mucosæ. The solitary lymphatic glands are considerably swollen, and their cellular elements tend to diffuse themselves through the surrounding tissue and to compress the glands in the immediate neighbourhood. With the exception of the signs of hyperæmia, and some slight degree of cell exudation, the submucous and muscular coats of the stomach do not exhibit any noticeable deviation from the normal.

Acute catarrh of the duodenum, arising from local irritation of the tissue by the products of food-fermentation, presents microscopic changes of a similar character to those just described. The superficial bloodvessels are congested, and the connective tissue of the villi, and that supporting the glands of Lieberkühn, is more or less densely infiltrated with small round cells. The ducts of the glands contain an excess of goblet cells, and their mouths are distended with mucus, epithelium, and blood corpuscles. The glands of Brunner, on the other hand, are seldom affected by the inflammatory process, nor does the submucous tissue suffer in the majority of cases. Superficial hæmorrhages are of frequent occurrence in the first portion of the duodenum, and occasionally terminate in ulceration. The solitary glands are also prone to undergo acute necrotic changes, and to be eventually converted into ulcers of the follicular type.

Considerable stress is usually laid upon spontaneous detachment of the epithelium from the surface of the intestine as a sign of acute inflammation; and some authors have sought to recognise a distinct variety of the disease under the title of 'desquamative catarrh.'[1]

But although it is true that the superficial cells are usually shed at an early stage of the disease, the phenomenon is by no means a constant one. In many cases not only does the epithelium persist for a considerable time, but the frequent discovery of shrunken and degenerated cells upon the free surface of a section appears to indicate that the columnar epithelium may occasionally pass through several stages of degeneration without being necessarily detached. On the other hand, the epithelial lining of the small intestine is extremely apt to fall off, as the result of post-mortem changes in the walls of the bowel ; and, indeed, I have seldom found the superficial epithelium to be intact when more than twenty-four hours had elapsed between the death of the patient and the removal of the tissues from the body. During the summer months post-mortem changes usually take place with great rapidity, and the columnar cells are often detached from their basement membrane within a few hours of death, and can be recognised in an apparently healthy state in the mucus contents of the gut. The epithelium is also frequently injured or displaced during the preparation of the tissue for the microscope, especially if the wall of the intestine is carelessly washed with tap water, immersed in too strong a solution of spirit, or cut with an indifferent knife. For these reasons I am inclined to attach but little importance to the existence or non-existence of the superficial epithelium in the absence of other histological appearances indicative of acute inflammation.

---

[1] Holt, *op. cit.*

The microscopic features of acute catarrh of the small intestine are essentially the same as those already described, namely, vascular injection, interstitial infiltration of round cells, and secondary changes in the epithelium of the tubular glands. In the case of the large intestine, examination with the microscope often reveals the existence of a vast number of minute follicular ulcers which were previously invisible to the naked eye. The coalescence of these minute abrasions, and the involvement of the larger masses of lymphoid tissue, produce those large ulcers the characters of which have been already detailed.

When death occurs rapidly, as in cases of so-called cholera infantum, the microscopic signs of disease are almost as trifling as those observed by the naked eye. The columnar epithelium is usually detached, the superficial bloodvessels slightly engorged, and minute hæmorrhages may be visible here and there in the substance of the mucous membrane. The solitary lymph follicles are enlarged, and a slight increase in the number of nuclei may be detected in the connective tissue of the villi and between the tubular glands.

By means of appropriate staining reagents, a few bacteria and cocci can often be discovered in the superficial portions of the tissue and in the capillary bloodvessels. In less acute cases the histological features of the various tissues closely approximate to those which have already been described when treating of the simple form of the acute catarrhal disease.

BACTERIOLOGY.

Although numerous efforts have been made to discover the exciting agent of the disease, the specific micro-organism has not as yet been isolated. The fact that the *Bacterium coli commune* often exists in preponderating numbers in the stools, while the injection of pure cultures of this bacillus into the general circulation of inferior animals gives rise to severe catarrh of the stomach and intestines, has induced certain writers to favour the assumption that this species is the one in request. But the bacterium in question is one of the normal inhabitants of the intestine from the earliest period of life, so that, in order to explain its sudden acquisition of injurious properties, it is necessary to admit either that its natural virulence is apt to undergo a spontaneous and remarkable intensification, or that admixture with some other and more virulent species is responsible for its increased activity. As a matter of fact, the coliform bacillus, which can be so readily isolated from the stools in cases of gastro-intestinal catarrh, possesses a comparatively low degree of virulence. Thus, in eight cases which I investigated with reference to this point, pure cultures of the bacillus made from the stools were found to be almost innocuous, since it usually required from 3 to 5 c.c. of a broth cultivation to produce serious symptoms in rabbits, while one-sixth of the dose of the same bacteria obtained from a healthy case was often followed by a fatal result. The *B. lactis aerogenes*, which is also frequently encountered, does not apparently possess any pathogenic properties. Among the members of the proteus series which are met with in the stools, Booker[1] was able to isolate one species, the injection of which into rats and rabbits was followed by death. Lesage[2] and Hayem[3] state that green diarrhœa in children is dependent upon the presence of a specific micro-organism, which, when injected in pure culture into the general circulation of an animal, appears within a short time in the duodenum, and gives rise to diarrhœa. These results, however, have not been confirmed (Jeffries, Baginsky), nor have I ever been able to isolate the chromogenic bacillus in cases of green diarrhœa. Baginsky[1] examined forty-three cases of summer diarrhœa, but failed to find any micro-organism of a specific character; while Booker,[2] who was able to isolate thirty-three forms of bacteria from cases of infantile diarrhœa, could not discover any constancy in the types which he observed.

---

[1] Quoted by Rotch, *op. cit.*

[2] *Le Bullet. Med.*, December 26, 1887.

[3] *Bulletin Géner. de Thérap.*, 1887, p. 441.

Tomkins[3] has described a form of stumpy bacillus which was constantly present in the walls of the intestine in cases which had succumbed during an epidemic of summer diarrhœa, and was also able to identify the same bacterium in the gas of the town sewers. According to Czerny and Moser,[4] acute gastro-enteritis is a result of a systemic infection, since in twelve out of fifteen cases of the disease which they examined they were able to find various micro-organisms in the blood (staphylococci, streptococci, bacilli), while in cases of simple dyspepsia the blood was sterile.

The weight of evidence appears to indicate that the symptoms of infective gastro-intestinal catarrh are dependent upon the absorption into the general circulation of certain organic poisons manufactured from the food by micro-organic activity. Where the symptoms develop within a short time after the administration of stale or sour milk, these poisons are in all probability already present in the food in a toxic dose, and in this connection it is interesting to observe that Vaughan[1] has been able to isolate a crystalline body (tyrotoxicon) from poisonous milk, which when given to animals produces symptoms allied to those of infantile cholera. Flügge[2] describes twelve different spore-bearing micro-organisms which he found in milk, many of which were capable of producing poisonous peptones from the casein. The milk cultures of three species were found by Lubert[3] to produce fatal diarrhœa when injected into the stomachs of young puppies. Duclaux,[4] on the other hand, considers that the lactic acid-forming bacilli in milk are directly responsible for the causation of epidemic diarrhœa.

---

[1] *Deut. Med. Woch.*, 1888, s. 391.

[2] *Trans. Amer. Pediat. Soc.*, i., 1889, p. 198.

[3] *Brit. Med. Journ.*, 1888, p. 417.

[4] *Jahrb. f. Kinderheilk.*, xxxviii., 1894, s. 487.

SYMPTOMS.

The symptoms which attend acute catarrh of the alimentary tract vary considerably in severity in different cases, being sometimes extremely acute, and leading to a fatal termination within a few hours, while in other instances they either subside spontaneously, or, gradually assuming a subacute or chronic character, run a protracted course. It is the usual custom to recognise three clinical varieties of the complaint, termed respectively the 'inflammatory,' 'choleraic,' and 'dysenteric,' according as the intestinal symptoms appear to resemble most closely those of simple inflammation of the bowel, of cholera, or of tropical dysentery. It is probable, however, that the first two varieties merely represent different degrees of severity of the same disease, while the so-called dysenteric form is dependent either upon follicular ulceration of the large intestine or upon a totally distinct disease characterized by croupous inflammation of the colon. I shall therefore depart from the orthodox methods of description, and merely deal with the clinical features of acute gastro-intestinal catarrh arising from any cause, pointing out, where necessary, the variations which occur in the severity of the symptoms in different cases.

---

[1] *Medical News*, June 9, 1888.

[2] *Zeitsch. f. Hygiene*, Bd. xvii., s. 272.

[3] *Ibid*, Bd. xxii., s. 1.

[4] *Annales de l'Institut Pasteur*, tome ix., No. 4.

Mode of Onset.—In the simple form of the disease the acute manifestations appear at first to be merely an exaggeration of those arising from the antecedent dyspepsia, and it is not until after the lapse of twenty-four hours, or even longer, that the abnormal severity and intractability of the symptoms indicate the existence of catarrh. The epidemic malady, on the other hand, either commences in an abrupt manner, or supervenes after a short period of general malaise, and develops so rapidly as to menace the life of the child within the course of a few hours. As a rule, the initial symptoms consist of vomiting and diarrhœa, but occasionally the disease is ushered in with acute delirium, shivering, high fever, or a series of epileptic fits.

Vomiting.—This symptom is almost invariably present at the commencement of an attack, although it varies in severity in different cases. The initial emesis affords temporary relief by removing from the stomach a large quantity of undigested food; but it is soon followed by severe retching, with occasional expulsion of a little glairy mucus and frothy saliva.

When the vomiting continues urgent, bile often regurgitates from the duodenum, and imparts a characteristic yellow or greenish tinge to the ejecta; while sometimes the rupture of a minute capillary vessel in the pharynx or stomach causes it to be tinged with blood. In the most acute and severe form of the disease the vomit tends to become thin and abundant, and closely resembles the rice-water evacuations from the intestine. Emesis can always be easily provoked by the ingestion of any form of nourishment, and even ice or cold water is apt to be rejected as soon as swallowed. With the progress of the complaint the vomiting gradually subsides, and in fatal cases it usually ceases as soon as symptoms of collapse manifest themselves.

Pain.—Pain in the abdomen is present in every case, and is responsible for the twisting and writhing movements of the body, as well as for the rigid flexion of the thighs and legs, by which the infant seeks to relieve the pressure exerted by the abdominal parietes upon the inflamed bowel.

From time to time the face assumes an agonized expression. accompanied by rotation of the eyeballs, and spasmodic contraction of the extremities. These various symptoms usually abate as soon as the diarrhœa becomes profuse, but they may recur upon each act of defæcation.

Diarrhœa.—A loose state of the bowels constitutes the main feature of the complaint, and in mild instances it is often the only objective phenomenon of any importance. In the majority of the cases the evacuations consist at first entirely of undigested food, which possesses an acid reaction and a sour smell. After a short time they tend to become more liquid, and are passed with noise and straining. The stools themselves vary greatly, both in colour and consistence, being at one time dark and feculent, while at another they are pultaceous or frothy, with a gray or greenish tint. As soon, however, as the solid contents of the bowel have been discharged, the motions generally become thin and watery, and often consist of a brown or dirty-green fluid charged with an intensely foul odour. Sometimes streaks of blood may be observed, or the stools may appear to consist entirely of blood-stained slime; while in children who suffer from worms large numbers of the parasites are eliminated at each action of the bowels. The more acute the attack, the more rapidly do the motions assume a watery character, and in the most severe cases the evacuations within a few hours may consist entirely of a green, gray, or colourless fluid, which leaves hardly any perceptible stain upon the napkins. When the disease has lasted for several days, and has occasioned follicular ulceration of the colon or rectum, the stools are reduced in quantity, though not in frequency, and the slimy material of which they are mainly composed is evacuated with much straining, and is apt to be acccompanied by prolapse of the bowel. Considerable stress is usually laid upon the frequency of the evacuations as an indication of the severity of an attack, a mild case being accompanied by three to seven motions in the course of the twenty-four hours, while those of a more acute character may exhibit twenty, or even more, in the same period of time. It must be remembered, however, that the deleterious effects of diarrhœa depend upon the drain of serous fluid from the body, and that consequently it is the presence of copious liquid stools which constitutes the chief danger to life, and not the frequent passage of small quantities of mucus, which is so frequently confused with true diarrhœa.

At the commencement of an attack the stools are always acid in reaction, and in most cases continue so throughout the whole course of the complaint. Even the green stools are usually acid, though to a less degree than normal. Microscopic examination reveals the presence of fragments of undigested casein and fat, along with a varying amount of epithelium, crystalline matter, blood-cells and bacteria. In the 'choleraic' form of the disease the motions soon lose their normal acidity, and become neutral, or even alkaline, in reaction, while the most careful examination fails to demonstrate anything of a more solid nature than a few epithelial cells and micro-organisms.

Almost every case is accompanied throughout by an elevation of the temperature of the body, although under ordinary conditions the fever is moderate in severity.[1] During the early part of the day the thermometer in the rectum may register 99° to 100·5° F.; but after noon the temperature begins to rise, and by 5 p.m. it may exceed 103° F. Occasionally severe fever accompanies the onset of the disease, and persists until diarrhœa sets in. Thus, in a case which I recently saw, the sudden development of vomiting, followed by convulsions and a temperature of 105·4° F., occasioned considerable anxiety, until the evacuation of several copious and liquid stools on the following day indicated the real nature of the complaint. In most cases the fever gradually declines with the approach of convalescence, but occasionally it persists for several days after the intestinal symptoms have disappeared. When a fatal termination is imminent, the internal temperature usually rises rapidly, and even continues to rise for a short time after death has taken place. An elevation of temperature also accompanies the development of broncho-pneumonia and other pulmonary complications, and hence in all cases where a sudden accession of fever occurs, a careful examination should be made of the whole body. In the 'choleraic' form of the disease a considerable degree of fever is usually present from the outset (103° to 105° F.), notwithstanding the fact that the surface of the body is usually cold and that symptoms of collapse are present. In these cases a sudden fall of temperature often precedes the cessation of the gastro-enteric phenomena, and is a sign of good omen ; but a sudden increase of the fever usually betokens the near approach of death.

---

[1] The thermometer should always be held either in the mouth or the rectum, as the temperature of the skin is often diminished in these cases.

In every case the *pulse* is abnormally quick, and tends to become weak and intermittent. In severe cases it is impossible to count the individual pulsations at the wrist, and with the advent of collapse the radial pulse may disappear altogether, while the heart-sounds become toneless, and finally inaudible.

From the outset the infant exhibits great restlessness and irritability, writhing in its cot or in its mother's arms, and giving vent to a constant succession of short cries, grunts, or moans. When pain is a prominent symptom, the child fails to sleep, and continues to cry incessantly for hours together, only ceasing when forced to do so from sheer exhaustion.

As the disease progresses, the voice becomes weak, and finally inaudible, though the sense of abdominal discomfort is still plainly expressed by frequent contortions of the face. At the commencement of an attack the cheeks are flushed, and the forehead and neck are often covered with sweat; but after a short time the face becomes pale, and the skin loses its natural elasticity and feels harsh and dry. The loss of fluid entailed by the vomiting and diarrhœa is responsible for the shrinkage of the tissues of the body which takes place after these symptoms have existed for a short time. The eyes appear to recede into their sockets, and a deep cleft appears between the globe and the rim of the orbit. At first the upper eyelids are often retracted, and the eyes consequently acquire a peculiar staring appearance; but with the progress of exhaustion the natural winking movements of the eyelids cease, the upper lids droop and obscure the greater portion of the cornea, while the lower lids become somewhat tumid, and present a blue-black coloration from venous engorgement. The naso-labial furrows deepen and extend, and the mouth becomes surrounded by a wrinkle, which is exaggerated by every contraction of the facial muscles. Elsewhere the superficial tissues exhibit a similar loss of elasticity, and the skin over the abdomen and inner surfaces of the arms and thighs appears wrinkled and flabby, and permits of being pinched up into loose folds which disappear with difficulty. Finally, the subcutaneous fat undergoes absorption, and the weight of the body rapidly diminishes. The state of the *tongue* depends to a great extent upon the condition of the stomach, being thickly furred when vomiting is a prominent symptom, but clean, red, and somewhat dry when diarrhœa constitutes the principal feature of the case. The *appetite* is always diminished; but the intense *thirst* from which the infant suffers incites it to swallow with avidity any form of nourishment in a liquid form. The constant irritation of the acid dejecta causes the skin around the anus to become reddened and excoriated; and in many cases the buttocks, as well as the inner sides of the thighs, present a shiny or glazed appearance; while small irritable papules occasionally develope over the affected parts, and by a subsequent process of ulceration, may give rise to circular sores of some depth.

After the disease has lasted for several hours or days, according to the severity of the attack, signs of exhaustion make their appearance: the infant ceases to cry, and becomes drowsy and apathetic; the face acquires a dusky or leaden colour, the eyes are sunken, the features pinched and withered, while the lips become livid, and surrounded by a zone of blue-black hue. The anterior fontanelle is deeply depressed, the tongue grows brown and dry, and patches of thrush make their appearance upon the mucous membrane of the mouth and palate. The surface of the body becomes cold, and the hands and feet blue, puffy, or œdematous. In most cases the temperature in the rectum undergoes a slight elevation; the pulse is quick, feeble, and irregular, and the breathing rapid and shallow.

PROGRESS AND TERMINATION.

From this state the child either gradually recovers or succumbs with the symptoms of profound collapse.

In the former case, the first sign of good omen is usually a decrease in the temperature of the body and cessation of the vomiting, the diarrhœa also becoming less frequent, while the stools exhibit traces of fæcal material. Occasionally the converse is observed, and an intestinal symptoms cease before the gastric. As soon as the child can retain nourishment in its stomach, the signs of collapse rapidly subside. The tissues gradually lose their shrunken appearance, the skin assumes a more healthy tint, and the pulse improves in volume, while it diminishes in frequency. Other symptoms of favourable import are to be observed in an increased secretion of the urine, and a substitution of an intelligent interest in its surroundings for the apathetic appearance which the infant had previously exhibited. From this time onward the child slowly but steadily improves, though it may be weeks before it completely recovers from the effects of its indisposition.

When reaction fails to occur, the infant passes into a state of profound collapse. The drowsiness increases, and finally merges into coma; the corneæ become cloudy, and the conjunctivæ exhibit a streaky vascular injection, while in many cases shreds of mucus collect over that portion of the eye which remains exposed between the half-closed lids. The pupils are usually contracted, and react tardily to light, but with the near approach of death they tend to become unequal, and finally widely dilated. The respiratory movements are quick, shallow, and irregular, and from time to time an expiratory effort is prolonged in the form of a sigh. The breath, as well as the tongue and mucous membrane of the mouth, feels quite cold, and the pulse at the wrist soon becomes lost, so that the only sign of continued circulation is to be found by auscultation of the heart. The fontanelle is greatly depressed, and the diminution in the quantity of the arachnoid fluid permits the bones of the skull to overlap along the sutures. The urine is suppressed. Vomiting usually ceases with the advent of collapse, and the diarrhœa either diminishes in severity or the stools are passed in the form of an incessant dribble. In some cases the internal temperature gradually declines, but in the epidemic form of the disease it usually undergoes a rapid elevation toward the end of life. Muscular twitchings are often observed, and not infrequently seizures of an epileptiform nature occur, and bring life to an abrupt termination. In severe cases, death takes place within a few hours of the onset of the stage of collapse, but in most instances the infant remains in a state of stupor for many hours, or even days, before it succumbs to gradual asthenia, or to hypostatic congestion of the lungs.

The duration of the disease varies according to the intensity of the symptoms and the previous state of the child's health. Among the richer classes of society, where children are well nourished and carefully attended, an ordinary attack of acute gastro-intestinal catarrh usually lasts from two to six days, and is often brought to a successful conclusion ; but among the poor inhabitants of large cities, the tendency to neglect the complaint in its early stages, combined with the inability to provide the care and nourishment requisite for the maintenance of life, causes the disease to be attended by an extremely heavy mortality ; and even in those cases where immediate danger is happily averted, the complaint is very apt to slide into a chronic state, and finally to cause death either by exhaustion, marasmus, or some intercurrent disorder.

In the most acute and severe form of the disease, which is often termed 'infantile cholera,' the various stages follow one another in rapid succession, and death often occurs within six to twenty-eight hours of the onset of the malady. Cases of this kind are, however, of comparatively rare occurrence; and I find that among 500 consecutive cases of acute gastro-intestinal catarrh which came under my care at the Evelina Hospital, only eleven, or about 2 per cent., could be placed in this category. Of this number, nine died and two recovered.

COMPLICATIONS.

Cerebral convulsions are observed in a large percentage of the cases at one time or another during the course of the complaint. In the early stages of the disease they are usually epileptiform in character, and tend to recur at short intervals. They most probably owe their origin to the absorption of organic products of bacterial activity from the alimentary canal. At a later period similar convulsions may arise from anæmia of the brain, consequent upon the profuse drain of fluid from the circulation; or spasmodic twitchings may occur in certain groups of muscles of the face or extremities. *Thrombosis* of the cerebral vessels occasionally takes place, and has been held responsible for the appearance of eclampsia in certain cases. Thus, an infant, aged sixteen months, which recently came under my charge for acute gastro-intestinal catarrh of five days' duration, was suddenly attacked with eclamptic seizures. affecting the left side of the body. The convulsions lasted for several hours, and finally disappeared, leaving the child in a comatose condition. The fingers of the left hand were then firmly clenched over the thumb, the fore-arm flexed, and the lower extremity on the affected side so rigidly extended that it seemed impossible to bend the joints without fracturing the bones. Shortly after the fits ceased the forehead and upper portion of the face became swollen, blue, and œdematous, and paresis of the right arm set in. The child never regained consciousness, and died within forty-eight hours of the initial seizure. Unfortunately, it was impossible to obtain a necropsy on this interesting case, but it is probable that the symptoms were due to thrombosis of the superior longitudinal sinus, involving some of the superficial cerebral veins of the right side, and to a lesser degree those of the left. Occasionally an attack of *tetany* supervenes during the later stages of the disease, but it is less common in the acute than in the chronic form of the complaint. The onset of the disease is preceded by severe vomiting or profuse diarrhœa, and the tonic spasm affects both arms and legs, although it is usually more pronounced on one side than the other. The superficial and deep reflexes are greatly exaggerated, and the urine is retained in the bladder. Attacks of this nature often persist with occasional intermissions for twenty-four to thirty-six hours, or even longer, at the end of which time the spasm suddenly disappears. This variety of secondary tetany appears to be less dangerous than that which accompanies chronic dilatation of the stomach in adult life,[1] for, of the five cases which I have observed, only one succumbed. In rare instances twitching of the facial muscles, rolling of the eyeballs, with rigidity of the arms or legs, are associated with suppression of the urinary secretion or with the elimination of only a small quantity of highly albuminous urine. These symptoms usually terminate rapidly in coma, and must therefore be regarded as phenomena of a uræmic nature.

Secondary inflammation of the respiratory organs is a frequent result of acute catarrh of the alimentary tract in infancy. *Bronchial catarrh* is particularly prevalent, and rhonchi or mucous rales can be detected in almost every case where the digestive disorder has lasted for more than a week. *Laryngitis* occurs in about 60 per cent. of all cases in which severe vomiting persists for more than twenty-four hours, and is responsible for the hoarse intonation of the voice which so often accompanies the later stages of the complaint. According to Lorr, the laryngeal affection arises by a process of extension from the œsophagus and stomach ; but this appears to be open to doubt. *Broncho-pneumonia* frequently complicates catarrh of the smaller tubes, especially in the infective form of the digestive disorder. Thus, I find that signs of bronchitis existed in no less than 87·3 per cent. of my cases which had lasted more than four days, while lobular consolidation of the lungs was encountered in nearly 37 per cent. of the entire number. This tendency to catarrhal pneumonia increases with the duration of the primary complaint, for, according to the observations of Holt,[1] nearly 50 per cent. of all cases of intestinal catarrh show signs of the pulmonary affection after the tenth day. The intimate connection which exists between inflammatory diseases of the lung and a similar morbid state of the digestive tract is also well shown by the researches of Miller,[2] who found that among 15,000 cases of pneumonia occurring in young infants nearly 75 per cent. were secondary to catarrh of the intestine. *Acute pleurisy* is only observed in a small proportion of the cases; but when it occurs the amount of effusion is usually considerable, and tends to become rapidly purulent. Kjellberg[3] was the first to draw attention to the existence of *parenchymatous nephritis* in cases of intestinal catarrh, no fewer than 47 per cent. of his fatal cases having exhibited evidence of renal mischief. The fact that the convoluted tubules of the kidney often present signs of inflammation after death from acute intestinal catarrh has been confirmed by Hirschsprung,[4] Epstein,[5] and others, although my own experience does not lead me to believe that it occurs as frequently as has been supposed. Thus, the presence of albumin in the urine was only noted in 17 per cent. of my cases of acute gastro-intestinal catarrh which came under treatment before the fifth day of the disease, and in no instance did the urine show more than a trace of it. In most cases where albumin is present, a few hyaline casts can be detected with the aid of the microscope; but I have never observed either blood-corpuscles or epithelial casts. The special symptoms which are supposed to accompany the renal complication comprise stiffness and œdema of the legs, loss of elasticity of the skin over the abdomen, a dry condition of the tongue, and constant attacks of retching and vomiting. The child exhibits great restlessness, and may be slightly convulsed from time to time.

---

[1] Author, *Trans. Clin. Soc.*, vol. xxviii., p. 13.

---

[1] Keating's 'Cyclop. Diseases of Children,' vol. iii., p. 61.

[2] *Wratch*, p. 352, 1892.

[3] *Journ. f. Kinderkrank.*, s. 275, 1870.

[4] *Jahr. f. Kinderheilk.*, s. 423, 1883.

[5] *Pädiatrische Arbeiten*, s. 303, 1890.

Among the intestinal complications of acute catarrh, *follicular ulceration* of the large intestine is the most frequent and important. This affection occurs in about 28 per cent. of all cases which last more than a week, and is responsible for the diarrhœa which so often persists after the more urgent symptoms of the complaint have subsided. The child experiences considerable griping pain in the abdomen, which is aggravated at each act of defæcation. The motions are passed at frequent intervals, and are accompanied by tenesmus. The stools vary in size from a drachm to an ounce and a half, or more, and consist almost entirely of thick mucus. Occasionally the evacuation is composed of bright blood, but in such cases it is usual to find that the mucous membrane of the rectum prolapses during the act of defæcation. These symptoms are often difficult to treat, and in weakly children may give rise to fatal exhaustion. It has already been shown that *intussusception* is frequently discovered after death, having apparently occurred immediately prior to the termination of life. In some cases, however, it takes place during the course of the disease, and is directly responsible for the fatal event. In such cases, I have often been struck by the insidious nature of the complaint, and by the absence of those symptoms which ordinarily portray its existence. Thus, the diarrhœa may continue, and the abdomen fail to exhibit any signs of distension for many hours, and it is only the continued presence of blood in the stools, or the accidental discovery of a tumour in the region of the colon, which affords any indication of the existence of this dangerous complication. *Ulcerative stomatitis* or *thrush* almost invariably make their appearance in severe cases; and in poorly-nourished children such *skin affections* as strophulus, urticaria, and eczema of the scalp, face, and ears, are particularly common. Erythema and excoriation of the skin of the anus and buttocks have already been noticed. *Sclerema* has been described as occurring occasionally in weakly subjects after an attack of cholera infantum. In this condition the skin is hard, white, and cold; the temperature of the body falls below normal, and the child dies from collapse.

Among the sequelæ of the disease, *acute miliary tuberculosis*, *chronic intestinal catarrh*, *marasmus*, and *tabes mesenterica*, are of considerable importance, and will be more fully described in the next chapter.

DIAGNOSIS.

The distinction between severe cases of intestinal dyspepsia and mild cases of catarrh of the digestive tract is often a matter of some difficulty. If, however, retching and vomiting continue after the residue of the last meal has been rejected, and especially if mucus or bile appears in the vomit, it is safe to conclude that the mucous membrane of the stomach is inflamed. Similarly, if the temperature of the body is slightly elevated toward evening, while the stools become thin and watery, and the child exhibits symptoms of collapse, it is certain that the intestine is also affected by the catarrhal process. Occasionally scarlatina, measles, or some other specific fever, is ushered in by vomiting or diarrhœa of sufficient severity to distract attention from the primary disorder. In such cases, however, the temperature usually rises to a point considerably above that which results from simple gastro-intestinal catarrh, and is constant rather than intermittent in type ; while the flushed appearance of the face, and the subsequent development of the characteristic eruption or some other important symptom, renders the differential diagnosis a matter of certainty. Typhoid fever occasionally commences in an abrupt manner with profuse diarrhœa and vomiting ; but this disease is rare in infancy, and after the first day or two runs a distinct course. A much more common error resides in the confusion of intestinal catarrh with that form of ulcerative colitis which occasionally attacks infants and young children who have been supplied with contaminated water, or exposed to the foul exhalations from refuse-heaps and sewers. The post-mortem appearances of this disease closely resemble those of tropical dysentery, and abscess of the liver has even been known to follow an attack. Nevertheless, there is no reason to suppose that the disease in question is really the same as the tropical complaint. Intussusception has also been mistaken for catarrhal enteritis. In this disease, however, the symptoms develop abruptly, and a considerable quantity of mucus and dark blood is usually voided by the bowel. Fever is invariably absent at the commencement, and the temperature seldom rises until distension of the belly or the presence of an abdominal tumour indicates the true nature of the complaint.

The distinction between the simple and infective forms of gastro-intestinal catarrh is seldom a matter of much difficulty. The former variety is almost entirely confined to hand-fed children who have usually suffered for a considerable time from the symptoms of gastric and intestinal dyspepsia. The disorder begins in an insidious manner, and is seldom accompanied by any degree of fever. The number of stools averages from four to seven daily, and they are characterized by the frequent changes in colour and consistence which they present, rather than by the thin and watery appearance which accompanies the more severe form of the disease. In the infective variety, on the other hand, the symptoms commence suddenly after a short spell of hot weather, and a large number of children are attacked simultaneously. The temperature rises at once, and the stools are passed at short intervals, and soon become watery and colourless, while the infant lapses into a condition of profound collapse.

PROGNOSIS.

Acute catarrh of the stomach and intestine in infancy is always fraught with danger to life, no matter how slight the symptoms may at first appear. Chronic malnutrition, or some previous disorder of the digestive organs, renders a child peculiarly vulnerable to the acute disease. For a somewhat similar reason, children who are attacked during the period of convalescence from one of the specific infectious fevers are extremely prone to fall victims to the complaint. A sudden onset, accompanied by convulsions, a high temperature, and profuse liquid diarrhœa, is usually indicative of a severe attack, while the early development of convulsions, stupor, or collapse are signs of evil omen. On the other hand, a steady decline of the fever, with a diminished frequency of the stools, and ability to retain nourishment in the stomach, may be regarded as favourable signs.

TREATMENT.

General.—The child must be confined to its cot from the commencement of the disease, and all unnecessary handling and movement avoided on account of its tendency to excite the peristaltic action of the intestines. The air in the bedroom should be maintained at a temperature of about 65° F., and free ventilation be secured by means of a fire or oil-lamp in the grate. The child should be clothed in a flannel shirt, and the bedclothes be sufficiently warm, but not oppressive. The temperature of the body must be carefully recorded every three or four hours, and should it remain constantly elevated above 101·5° F., or at any time rise above 102° F., some means should be employed to reduce the fever. As a rule, sponging the body with tepid water is quite sufficient to procure the requisite reduction of the temperature; but should the operation prove distasteful to the child or induce shivering, hot water (110° F.) may be used with an equally beneficial result. When extreme restlessness and persistent insomnia are associated with severe pyrexia, the application of a wet pack every few hours exerts a soothing influence upon the nervous system, in addition to its antipyretic effect, and as soon as the initial shock of the cold application has passed off, the infant will often cease its distressing cries and fall into a refreshing slumber. It is rare that the degree of fever proves so severe as to necessitate the employment of the bath, although many Continental physicians prefer this antipyretic method to any other. Should, however, the fever remain uninfluenced by milder measures, or the temperature rise above 104·5° F., and prove intractable, the child should be immediately immersed in water at a temperature of 100° F., which, by the addition of cold water or pieces of ice, is then cautiously reduced to 80-75° F. It is wise to administer a few drops of brandy or a little white wine whey after each immersion, in order to counteract the shock which often follows a sudden reduction of the body temperature. After each immersion the child should be quickly dried and wrapped in a blanket, while the bath is repeated whenever the temperature tends to regain its former height.

When abdominal pain constitutes a prominent symptom of the case, considerable relief can be afforded by the application of hot poultices, turpentine stupes, or a mustard leaf, to the epigastrium ; but the use of liniments, combined with active manipulation of the abdomen, should be avoided. As soon as symptoms of collapse manifest themselves, the infant should be enveloped in cotton-wool or a warm blanket, and bottles filled with hot water and covered with flannel placed at the feet and along the sides of the body. At the same time some form of diffusible stimulant, such as a few drops of brandy, sal volatile, or spirits of camphor, diluted with water, or a teaspoonful of champagne or white wine whey, may be given at short intervals by the mouth. If these measures fail, the child should either be placed in a hot bath containing mustard (two tablespoonfuls to the gallon), and energetic friction applied to the extremities, or some stimulating liniment, like those of turpentine or camphor, may be rubbed upon the chest and limbs. In cases of severe cardiac failure, accompanied by coldness and cyanosis of the skin, a mustard leaf or a flannel wrung out of hot water and applied to the precordium, combined with the subcutaneous injection of ether or brandy, are valuable stimulants to the action of the heart. The injection of cold water into the rectum, and of saline solution into the subcutaneous tissues, have also been recommended for a similar purpose, but they are seldom of any real value. Transfusion is sometimes performed with success in acute cases associated with sudden and severe collapse, but in most cases the improvement which follows the operation is only transient, and the infant soon dies from a recurrence of its former symptoms.

Lavage of the stomach has been highly recommended by many authorities for the treatment of persistent retching and vomiting arising from gastric catarrh (Demme, Epstein, Siebert), either simple warm water or a dilute solution of boracic acid (5 per cent.) being employed for this purpose. In certain cases the employment of the douche is attended by success, and the infant is enabled to retain small quantities of nourishment. More frequently, however, the effects of the lavage are disappointing, and the vomiting recurs with increased severity. Good results are sometimes obtained from the employment of a soft tube for the purpose of injecting food into the stomach. When fed in this manner, it is often surprising how readily an infant will retain several ounces of liquid nourishment, when the administration of only a few drops by the mouth is followed immediately by vomiting.

Irrigation of the large intestine in cases of acute intestinal catarrh has been practised with success both in Germany and America. Warm saline solution (sixty grains to the pint) is introduced through a soft tube inserted high up into the rectum, and the operation continued until the fluid returns clear and free from fæcal matter. The ascent of the fluid is aided by elevation of the buttocks and gentle massage of the colon in the direction of the cæcum. This method may be employed once or twice each day.

It is extremely important to ensure the immediate and complete disinfection of any linen or wearing apparel which may have become accidentally soiled by the vomit or alvine discharges. The napkins should therefore be changed as often as they become wet, and at once immersed in a solution of carbolic acid (one in forty) before being washed clean on the premises, while all infected blankets and bedding ought to be burnt. The utmost care should be taken to prevent inflammation of the skin about the anus and buttocks arising from contact with the dejecta, and with this object the parts should be frequently cleansed with warm water, and afterwards anointed with vaseline, thymol jelly, or boracic ointment.

As soon as the immediate symptoms of the disease have subsided, the child should be sent into the fresh air in a perambulator, care being taken, of course, to prevent fatigue or exposure to cold. When convalescence has set in, a change of air is imperative, especially if the patient lives in a large town; and even in subacute forms of the disease the prompt removal of the child to the country or seaside is often followed by a rapid improvement.

Dietetic.—In all cases the question of diet requires immediate attention. Owing to the suspension of the functional activity of the digestive organs which occurs as the result of acute catarrh, all articles of food which require to be digested before they can be assimilated must be prohibited, and only those forms of liquid nourishment administered which throw the least amount of strain upon the digestive organs. In the early stages of the disease every kind of food, especially milk, should be prohibited entirely for eight to twelve hours, and only a small quantity of barley, rice, or iced water be allowed from time to time, in order to assuage the thirst. As soon as the intolerance of the stomach has subsided, some bland form of nourishment may be cautiously tried. If the infant is a suckling, it may be put to the breast for a short time, or the mammary secretion may be artificially withdrawn and administered from a spoon. Should the experiment prove successful, the same quantity of food may be allowed after the lapse of an hour, and the child may subsequently be permitted to take a moderate meal from the breast every three or four hours. If the infant has only recently been weaned, it is wise to have recourse once more to the breast, or, if this is impossible, to procure a reliable wet-nurse. In the case of older children, or in those which have been artificially fed, all food should be prohibited by the mouth for at least six hours, and the return to a milk diet only permitted when the irritability of the stomach has been completely allayed. Thus, as soon as the more urgent symptoms have abated, the infant may be allowed to partake from time to time of small quantities of *cold* whey and barley-water, albumin-water (R. 4), or cold veal or chicken tea (R. 9), while at a later stage small doses of raw meat-juice, koumiss deprived of its gas, thin gruel (R. 2), kéfir, or mutton broth, may be employed. Beef-tea is also recommended at this period of the complaint, but it often appears to excite the peristaltic movements of the intestine, and to increase the diarrhœa. If the child continues to thrive, a cautious trial may now be made of milk, which, after being sterilized, should be mixed with at least three times its volume of barley-water, and administered in doses not larger than half an ounce at a time. Should the ingestion of the milk be followed by flatulence, retching, or the appearance of curd in the stools, it must either be withheld again for a time, or peptonized and flavoured with vanilla or lemon to render it more palatable. In some cases a cream mixture (p. 41) agrees better than diluted milk, or whey thickened with a small quantity of malted food or predigested gruel (p. 54) is retained when other forms of food are rejected. Gradually and with caution the amount of milk is increased, until at last the child is able to take the full allowance suitable to its age. Even when the stage of convalescence is well advanced, it is advisable to restrict the infant to a milk diet, with or without Mellin's food, and to avoid the use of unmalted foods for some time. It occasionally happens that the retching continues severe even after all food has been withheld for many hours, and it is consequently a question whether the administration of nourishment by the mouth will not further increase the vomiting. In such cases the excessive irritability of the stomach is often dependent upon exhaustion of the nervous system, and can be easily allayed by the addition of a teaspoonful of white wine whey, a few drops of brandy, or a little iced champagne, to the broth or other form of nourishment which is judged worthy of trial. In the most severe cases it is necessary to administer nutrient enemata (R. 8) combined with opium.

Medicinal.—There is probably no disease known in the science of medicine which affords greater scope for the display of ingenuity in the writing of prescriptions than the so-called 'infantile diarrhœa.' Hardly a week passes without the appearance in some medical periodical either of some new combination of old drugs which in the hands of its inventor has been attended by conspicuous success, or of some new remedy which, from the beneficial effects it produced in a certain number of cases, is lauded as an absolute specific for the complaint. Unfortunately, more extended experience has invariably proved that, however useful each of these various remedies may be in certain cases, there is not one which can be absolutely relied upon; and consequently the practitioner finds himself face to face with a monumental pile of drugs, each of which is highly recommended, but not a single one guaranteed. The result is that he soon becomes wearied with haphazard medication, and finally adopts some form of routine treatment which his experience indicates is as good as any other. This is, after all, only a natural outcome of the teaching which directs sole attention to the diarrhœa, while it ignores the disease of which it is merely a symptom, and includes under the absurd title of 'diarrhœa' a number of distinct diseases, each of which requires a separate treatment. Since there exists no justification for regarding vomiting, diarrhœa, or any other kindred symptom of disordered digestion, in the light of a clinical entity, I shall discard the usual custom of enumerating the various drugs which have been found of use in the treatment of the complaint, and confine myself to a brief description of the chief indications for the employment of medicinal remedies at different stages of the disease.

Our present knowledge concerning the etiology of acute gastro-intestinal catarrh indicates that in the vast majority of cases the morbid changes in the alimentary tract are the result, either directly or indirectly, of micro-organic activity. In many instances the severity of the symptoms exhibits a direct relationship to the degree of inflammation, and in these cases it is probable that the catarrhal process arises from the irritation of the mucous membrane by the chemical products of fermentation. In other and happily rarer instances, the catarrh appears to be quite a subsidiary phenomenon to the general infection of the system, and the vomiting and diarrhœa owe their origin primarily to a local vaso-motor disturbance induced in the alimentary canal by the presence of organic poisons in the circulation. It is obvious, therefore, that in either case the only true curative treatment for the disease is by the adoption of those measures which secure the destruction and elimination of the bacteria and their products.

The vomiting and diarrhœa which usually accompany the early stages of the complaint are of distinct value to the organism; they constitute, in fact, the first line of defence devised by Nature against noxious materials which find an entrance to the body through the digestive canal; and it is only when they persist after their work has been completed that they prove a curse rather than a blessing, as in the case of every other agency which gets beyond control. The first indication, therefore, in the treatment of the disease is to assist the alimentary canal to rid itself of those elements of danger which it contains. In order to ascertain the necessity for action, both the vomit and the stools should be submitted to a careful inspection. Owing to the undeveloped state of the stomach, the first act of emesis usually empties the organ, so that it is seldom any need arises for the employment of an emetic. If however, the breath smells sour, and masses of curd or fermenting particles of food continue to be rejected, a full dose of the wine of ipecacuanha (one drachm), sulphate of zinc (three to ten grains), sulphate of copper (half to one grain), may be given, or the stomach-tube may be used with advantage. On the other hand, the presence in the stools of undigested food, of yeast-like material, or small lumps of fæcal matter, indicates that the intestines still contain the sources of irritation, and means should consequently be taken to secure their expulsion. With this object, recourse may be had to some simple purgative, non-irritant in its action, and at the same time reliable in its effects.

As a rule, castor-oil meets these several requirements best, and two teaspoonfuls or more may be given at once. In young infants the repeated use of small doses of the oil is often attended by better results than the employment of a single full dose, and it is probably for this reason that the mistura olei ricini occupies so important a place in the pharmacopœia of every children's hospital (F. 1). Some physicians prefer to clear out the bowel by means of a saline draught (F. 1), others recommend a mixture containing rhubarb, aloes, or senna (F. 1) for a similar purpose; while others, again, place the greatest reliance upon the use of calomel. Personally, I have found the best results to attend a full dose of castor-oil, followed by the use of the castor-oil mixture every three hours, combined with total abstention from food; but whichever method is employed, there can be no doubt that the majority of cases of acute gastro-intestinal catarrh, if seen at an early stage and treated in the manner described, make a rapid and complete recovery.

Having thus procured the expulsion of most of the products of fermentation, as well as of the materials upon which the bacteria thrive, it is necessary to promote the asepticity of the digestive tract, and to allay the inflammatory process which has been excited. The second indication, therefore, in the treatment of the disease is to administer some drug or combination of drugs which possesses the power of inhibiting bacterial growth in the stomach and intestine. And here I would venture to draw attention to the important distinction which exists between the theory and practice of gastro-intestinal antisepsis. Experimental research has brought to light a large number of drugs which are endowed with powerful antiseptic properties, and it has been hastily assumed that each of these substances has a therapeutic value in direct proportion to its germicidal powers. This, however, is a mistake. The alimentary canal of a human being is something more than a test-tube; it is a vital organ endowed with the power of absorbing not only the poisonous products of the bacteria, but also most of the substances which are introduced to destroy them. In addition to this, the various secretions which are continually poured into the digestive tract are capable of altering the chemical composition of almost every substance introduced into it, in some cases converting a comparatively innocuous drug into an active poison, in others destroying the antiseptic properties of the original compound. Lastly, cognizance has been taken of the idiosyncrasies of various persons toward different drugs, and of the solubility, taste, and other physical properties which characterize the several remedial agents. These questions are of extreme importance in the treatment of infants and children in whom the wholesale methods of medication are at once both difficult and dangerous. The problem of intestinal antisepsis consequently resolves itself into this: with what drugs can we obtain the maximum antiseptic results with the minimum amount of discomfort or danger to the patient? Before venturing upon an answer to this important question, I would offer a few remarks concerning the general properties of the antiseptic remedies which we have at our disposal.

Calomel is an excellent example of that class of remedies which owe their antiseptic properties to a change in their chemical constitution occurring after owing to the fact that it undergoes decomposition in the bowel with the liberation of free iodine. It is, however, extremely difficult to administer to children on account of its pungent smell, and of late years it has been quite superseded by the aforementioned remedies.

Resorcine (metadioxybenzolum) is a crystalline powder, freely soluble in water, and possessed of a harsh, sweet taste. It is a cheap and powerful antiseptic, and devoid of poisonous properties when given in ordinary doses (two to five grains).

Carbolic acid, *creasote* and *perchloride of mercury* are all powerful germicides, but their value in diseases of infancy is somewhat impaired by their unpleasant taste and poisonous properties. Carbolic acid may be given either in the form of the pure drug, of which one minim can be administered well diluted with water every hour, or of the glycerine preparation (B.P., minims 2—5) in combination with an alkali or subnitrate of bismuth. Creasote is usually prescribed in half-minim doses suspended in some sweet emulsion, but is greatly inferior in value to carbolic acid. Infants tolerate a comparatively large dose of perchloride of mercury, so that it is possible to administer a teaspoonful of the solution (B.P.) every few hours without producing any bad effects. As an intestinal antiseptic it is less reliable than the insoluble preparations of mercury.

During the last four years I have tried these various antiseptic remedies in more than 500 cases of disordered digestion in infancy associated with diarrhœa, and although the adoption of the principle of intestinal antisepsis has been attended by brilliant results, I have been unable to convince myself that all the drugs in question were equally beneficial, or that the exhibition of the same substance in different cases was invariably attended by the same success. I will therefore offer a brief outline of the method of treatment which in my own hands has been followed by the best results. It has already been stated that in the majority of cases of moderate severity the administration of castor-oil at the commencement of the attack usually cuts short the disease by removing at once both the bacteria and their products from the bowel. This beneficial effect can often be enhanced by the addition of thirty to sixty minims of the solution of the perchloride of mercury, or a few grains of resorcine, to each dose of the castor-oil mixture. By this simple procedure many cases which at first resist the action of the oil rapidly lose their watery stools, and soon become convalescent. If, however, the diarrhœa continues in spite of the treatment, or, when first seen by the medical man, the case presents numerous thin, liquid stools, the employment of calomel is usually found to be followed by better results. For this purpose one-eighth to a quarter of a grain of the mercurial salt is administered every four hours. When given in this manner, calomel proves itself one of the most reliable drugs we possess for the treatment of acute gastro-intestinal catarrh, the vomiting and diarrhœa often subsiding under its influence within thirty-six hours.

Should the stools still continue frequent and loose, it is probable that the catarrhal process in the mucous membrane has reached an advanced stage, and requires the exhibition of sedatives in addition to the antiseptic remedies. It is in this condition that the subnitrate of bismuth is of such value, especially when combined with some non-irritant antiseptic like resorcine; but in order to be effective the drug must be given in doses of at least ten grains every two or three hours (F. 3). The usual habit of prescribing two or three grains for a dose is quite useless, and merely waste of valuable time. It is best to suspend the salt in chloroform-water, and to avoid the use of tragacanth or other viscid media. The first effect of the treatment is usually observed after the fourth dose, when the stools commence to decrease in frequency, and to present a more solid appearance. At the end of the second day the diarrhœa has usually ceased, and is sometimes replaced by obstinate constipation. The medicine should always be discontinued in a cautious manner, for it is often observed that the intestinal flux recurs if the treatment has been hastily abandoned. Salol, and the salicylates of strontium, lime, and bismuth, are also valuable at this stage of the complaint, but I have never been able to convince myself that they possess any advantages over the bismuth and resorcine mixture. When the diarrhœa has already persisted for several days, and the child exhibits symptoms of collapse, it is usually advisable to prescribe a small quantity of opium with one of the aforementioned remedies. Thus, a combination of Dover's powder (one-sixth to a quarter of a grain) with an equal quantity of calomel or gray powder is often extremely valuable at this period of the complaint (F. 3), or a similar quantity of the opiate may be added to the bismuth mixture with advantage. Opium is also invaluable in cases of a subacute nature where the passage of small mucoid stools is attended by tenesmus. For this purpose, Dover's powder (one-eighth to a quarter of a grain), or the tincture (one to two minims) or the extract (one-thirtieth to one-twelfth of a grain), is the most serviceable, though occasionally the compound tincture of camphor (three to seven minims), nepenthe (one to two minims), or the aromatic preparation of chalk with opium (five to eight grains), is to be preferred, as less liable to disturb the digestion. If the straining continues in spite of opium, and especially if prolapse of the rectum occurs after the passage of a stool, a combination of powdered ipecacuanha, carbonate of bismuth, prepared chalk, and Dover's powder is often attended by marked success (F. 2).

Occasionally this form of spurious diarrhœa is accompanied by severe vomiting, which, from the exhaustion it produces, greatly increases the gravity of the case. Under these circumstances, it will be found that one or two minims of the wine of ipecacuanha with half a minim of the tincture of opium every hour is more rapidly efficacious than anything else. Some authorities recommend antimonial wine for this purpose, but personally I have not found it so useful as ipecacuanha. In severe cases, where prolapse of the rectum occurs at each call to stool, and occasions great distress and considerable loss of blood, hot fomentations sprinkled with laudanum should be constantly applied to the perinæum, and, after the prolapse has been reduced, an enema composed of half an ounce of thin starch, with two to four minims of tincture of opium, and three grains of powdered ipecacuanha, may be carefully injected into the rectum.

Although the majority of the cases of acute gastro-intestinal catarrh are susceptible of rapid improvement when treated upon these principles, there yet remains to be considered that acute and formidable variety of the infective disease which has received the ominous name of 'infantile cholera.' In these cases the bacterial products are already present in the general circulation in a toxic dose, so that if it were possible to stay the further production of the poison by the administration of antiseptics, we yet lack the power of neutralizing that portion of it which is responsible for the immediate symptoms of the complaint. It is probably for this reason that most of the cases defy the most active medicinal treatment, and often die of respiratory or cardiac failure even after the cessation of the vomiting and diarrhœa.

There is little to be added to what has already been said respecting the general and dietetic treatment of the disease. All milk must be prohibited, and equal parts of cold whey and barley-water, or small quantities of chicken broth or meat-juice, should constitute the sole nourishment as long as the vomiting persists. Henoch recommended the subcutaneous injection of a 2 per cent. solution of common salt during the period of collapse, but the improvement which follows the adoption of this procedure is usually only of a transitory nature. The intense thirst must be combated by the free administration of iced water, and such stimulants as brandy, ether, ammonia, or champagne should be given as soon as failure of the heart becomes apparent.

With regard to the use of drugs in these cases, it is admitted by all that at present we possess no remedy of any certain value. The use of astringent or sedative drugs is only attended by an increase of the vomiting, while the employment of rectal injections is rendered useless by the frequency and severity of the diarrhœa. At an early stage the hypodermic injection of morphine ($\frac{1}{30}$ of a grain), with or without atropine ($\frac{1}{200}$ of a grain), is attended by better results than any other line of treatment, the dose being repeated at the end of one or two hours if the symptoms show no signs of abatement; but if collapse is already a prominent feature of the case, or the infant exhibits signs of stupor, morphine often does more harm than good, by increasing the tendency to pulmonary congestion. As soon as the vomiting subsides, soluble antiseptics, such as carbolic acid, creasote, tincture of iodine, or resorcine, may be given in full doses at short intervals.

During the convalescent period the digestive powers of the stomach should be increased by the judicious use of dilute hydrochloric acid in combination with pepsine, while the general health may be improved by the administration of such tonic remedies as iron, nux vomica, gentian, and afterwards of cod-liver oil.

CHAPTER V.

CHRONIC GASTRO-INTESTINAL CATARRH IN INFANTS.

(CHRONIC VOMITING—CHRONIC INFLAMMATORY DIARRHŒA—MARASMUS—INFANTILE ATROPHY—ATHREPSIA).

MANY terms have been employed at different times to describe a chronic disease of infancy characterized by vomiting, diarrhœa, or some other symptom of disordered digestion, and attended by gradual wasting of the tissues of the body. As a rule, the choice of nomenclature has been determined by the predominance of some particular symptom of the complaint, so that we find such apparently diverse terms as 'chronic vomiting,' 'inflammatory diarrhœa,' 'athrepsia,' and 'marasmus,' applied to the same disease, according as one or other of its symptoms happens to be most prominent. But however widely the various cases may differ among themselves in their clinical aspect, they all present one common feature after death, for in every instance the signs of chronic inflammation of the stomach and intestine, with more or less atrophy of the mucous membrane of the alimentary canal, can be demonstrated with the microscope. It appears, therefore, to be justifiable, as well as expedient, to group all the cases which exhibit this primary lesion into one class, and to designate the disease 'chronic gastro-intestinal catarrh.'

ETIOLOGY.

Chronic catarrh of the stomach and intestine is predisposed to by the same conditions which render children vulnerable to the acute form of the disease. Thus, congenital syphilis, rickets, tuberculosis, anæmia, and malnutrition, all favour the chronicity of the digestive disorder when once it has been excited; while overcrowding, defective ventilation, deficient or deleterious foods, or a previous attack of intestinal catarrh, eminently predispose to the acquisition of the more enduring form. Among the exciting causes of the disease, the habitual administration of unsuitable food, exposure to cold, or an attack of an acute specific fever are of the first importance. Chronic gastro-intestinal catarrh in infancy must consequently be regarded as essentially a preventible disease, inasmuch as the chief factors in its causation consist of a constant neglect either of the elementary laws which govern the diet and hygiene of early life, or of those minor disorders of the digestive system which usually precede the development of the complaint. With these facts in mind, it is hardly a matter for wonder that the disease is so disproportionately common among the poor inhabitants of large cities, whose children are exposed not only to the dangers arising from insufficient nourishment, but also to those engendered by the ignorant and superstitious practices current among all uneducated persons. Such people invariably cherish the delusion that palatable substances and food are synonymous terms, and it is impossible to convince them that the mere introduction of food into the stomach is quite valueless for the purposes of nutrition, unless the infant is capable of digesting and assimilating it.

Some of the worst cases of the disease occur among children who are put out to nurse during the day with some obliging neighbour who undertakes, for a small consideration, to provide both home and food for infants during the absence of the mothers at their work. Many of these 'nurses' appear to specialize in illegitimate children, and the remarkable regularity with which they bring their charges to the hospital in a practically moribund condition must convince every physician that an alteration in the law relating to the signing of death certificates in out-patient practice would materially lessen the yearly death-roll from so-called 'marasmus.'

Among 1,000 consecutive cases of disease in children which have come under my notice at the Evelina Hospital, 172, or 17·2 per cent., were found to be suffering from gastro-intestinal catarrh, the symptoms of which had existed for more than a month. From this number I have taken, without selection, the notes of 100 cases of the disease, and from an analysis of these I shall venture to offer a few remarks. In only 8 instances out of the entire number were the infants fed entirely upon the breast, the remaining 92 receiving either cow's milk or some kind of farinaceous food. These figures tally very closely with those which have already been offered regarding the relative frequency of dyspepsia among sucklings and hand-fed children respectively, and emphasize in a most striking manner the influence of diet in the causation of the chronic inflammatory ase. In 11 cases out of the entire number the plaint was stated to have followed immediately upon an attack of some specific infectious fever, of which pertussis, measles and scarlatina were the most important. Upon this point the observations of Rilliet and Barthez[1] are very striking, since they show that, among 140 cases of chronic diarrhœa in children, 27 were preceded by pneumonia, 37 by measles, 17 by small-pox, scarlatina, or enteric fever, and 29 by either angina, pleurisy, bronchitis or croup. With regard to the importance of exposure to cold as an exciting cause of the disease, I can offer no reliable statistics, since in out-patient practice it is impossible to discriminate between the influence of atmospheric conditions and the effects of those dietetic errors which constitute such an extremely common and important factor in the production of all diseases of infancy. There can be no doubt, however, that an attack of acute gastric or intestinal catarrh can be excited in the first instance by exposure to cold, and afterwards converted into a subacute or chronic form by the influence of one of the above-mentioned causes. The various ages at which chronic gastro-intestinal catarrh is most commonly contracted is a matter of some importance, and the cases at my disposal allow of arrangement in the following manner:

Age of Infant at Commencement of Disease.	No. of Cases.	Breast-fed.	Breast and Artificial Foods.	Artificial Foods only.
1-4 months	34	1	1	32
4-8 „	10	1	7	2
8-12 „	17	3	4	10
12-18 „	26	2	0	24
18-24 „	13	1	0	12
	100	8	12	80

---

[1] 'Trait. des Malad. de l'enfance,' ii., p. 346.

These figures present several points of interest. It will be seen at once that the disease is most common within the first four months of life, since no fewer than 34 per cent. of the cases occurred in infants of this age. During the next four months the cases only represent 10 per cent. of the entire number ; but from the eighth to the eighteenth month the susceptibility to the disease appears to increase, for we find that 43 per cent. of all the cases which came under observation were infants between eight and eighteen months old. After the eighteenth month the complaint becomes comparatively rare, and is seldom encountered after the third year of life This curious fluctuation in the severity of the disease at different periods of infantile life can only be explained by an examination of the kinds of food which were employed in the various cases prior to the onset of the inflammatory disease. Thus, among the 34 cases occurring in children less than four months old, 33, or 97 per cent., were habitually supplied with some form of artificial food, and only in one instance was the child fed exclusively upon the breast. In every case the infant had appeared to be perfectly healthy at birth, but within a short time had commenced to exhibit the symptoms of chronic dyspepsia, or to suffer from attacks of subacute catarrh of the stomach and intestine, accompanied by gradual wasting of the tissues of the body. It is probable, therefore, that the chief cause of chronic gastro-intestinal catarrh at this period resides in the injudicious administration of artificial foods in lieu of the maternal milk.

The comparative immunity from the disease which is apparent between the fourth and ninth months of life is probably due to the fact that at this period the majority of the children are either nourished entirely upon the breast, or, having escaped the initial dangers of artificial feeding, are able to take the bottle with impunity. But about the ninth month the common custom of supplementing the breast milk with biscuits, cornflour, or some other variety of farinaceous food, at once exposes the infant to the dangers arising from the administration of artificial foods, and consequently the disease once more becomes prevalent until the period when milk is finally discarded for the mixed diet of adult life. Among the wealthier classes, on the other hand, mothers who nurse their children are in the habit of employing some form of artificial food about the fourth month, so that, had these statistics been compiled from observations made in private practice, it is probable that the disease would have appeared to be more rife within the first six months than at any other time.

MORBID ANATOMY.

As in the case of every other disease of the digestive canal, the naked-eye appearances presented by the tissues after death are usually quite insignificant when contrasted with the violence of the symptoms observed during life. The stomach and small intestine are usually somewhat distended with gas, and occasionally the transverse colon and sigmoid flexure are also much inflated. The stomach invariably shows signs of dilatation, the exact degree of enlargement varying directly with the duration of the catarrhal disease and the amount of organic change which has taken place in the coats of the organ. As a rule, the lower border of the viscus seldom extends more than a finger's breadth below the level of the umbilicus, that enormous degree of dilatation which so often accompanies the chronic gastric catarrh of pulmonary tuberculosis being never observed.[1] When the stomach of an infant becomes dilated owing to weakness of its muscular walls, the shape it assumes differs from that which is observed under similar conditions in adult life. In the majority of cases, it still maintains its cylindrical form, owing to the cardiac and pyloric portions of the organ participating equally in the dilatation. Occasionally, however, the fundus is greatly distended, while the pyloric region still preserves its natural contour, and in such cases the lower end of the œsophagus is usually dilated, and appears to pass imperceptibly into the upper border of the stomach. Some writers have described a form of hour-glass dilatation of the stomach in infancy, but this variety I have never observed. In all cases where the stomach is dilated, the increased weight of the organ drags the pylorus downwards and inwards toward the median line of the abdomen, thereby causing it to describe an arc of a circle, the centre of which is situated at the œsophageal opening of the diaphragm.

Upon opening the organ and removing the thick layer of mucus which adheres to its inner surface, the mucous membrane is found to present a dead-white, opaque appearance, and to be more firmly adherent to the subjacent layers of tissue than under normal circumstances. Post-mortem digestion is rarely encountered. In long-standing cases, where the body has undergone great emaciation, the coats of the stomach and intestine are sometimes so extremely thin and transparent as to closely resemble tissue-paper. Vascular injection is usually absent, but should an attack of subacute catarrh have occurred shortly before death, the surface may exhibit a patchy or arborescent form of congestion. In very chronic cases the mucous membrane in the pyloric region sometimes presents a rudimentary form of mammillation, being mapped out into small polygonal areas of thickened and opaque tissue. In other instances, the secretory membrane exhibits a brownish or slate-coloured tinge as a result of chronic congestion, or minute extravasations of blood may be observed in the cardiac region of the organ or in the vicinity of the lesser curvature. Hæmorrhagic erosions are also frequently encountered in the cardiac extremity of the stomach, but the follicular form of ulceration due to disease of the solitary glands is rare, unless the primary complaint has been complicated with tuberculosis. Parrot[1] and other writers have described aphthous patches upon the mucous membrane similar to those which occur in the mouth, as well as the occasional formation of a false membrane like that observed in cases of diphtheria of the stomach. The former condition is, I believe, extremely rare; the latter I have never observed in simple cases of chronic gastric catarrh.

---

[1] Author, 'The Dyspepsia of Phthisis,' 1894, p. 2.

The small intestine seldom exhibits any more decided appearances of disease than those already noted. Peyer's patches are apt to be enlarged in the early stages of the complaint, and often present signs of congestion, or even minute hæmorrhages. Ulceration seldom occurs, but as the disease progresses an atrophy of the lymphoid tissue takes place. The lower portion of the ileum, within an inch or two of the ileo-cæcal valve, is occasionally the seat of an irregular form of ulceration, superficial in character, and not infrequently multiple. The mucous membrane in the immediate neighbourhood of the disease is thickened and pigmented from chronic inflammation.

---

[1] 'Athrepsie,' Paris, 1877, p. 241.

In all cases of chronic intestinal catarrh, the most characteristic phenomena are to be found in the larger bowel, especially near its lower end. Here thickening and adhesion of the mucous membrane to the muscular tissue is the rule, and in most cases the superficial aspect of the inner coat presents a mottled appearance, owing to the presence of an immense number of minute points of pigmentation. In the transverse colon and cæcum this feature is sometimes so marked that areas of the mucous membrane several inches in extent exhibit a uniform slate-coloured tinge. This condition is quite distinct from the superficial staining which results from the long-continued use of bismuth or arsenic. In about 62 per cent. of the cases where the disease has existed for more than four weeks, the large intestine is affected with follicular ulceration, the severity of which varies with the degree and duration of the catarrhal process. In mild cases, the inner surface of the colon and rectum exhibits numerous small pits, separated from one another by patches of congested tissue. In more severe instances, however, necrosis of the solitary follicles occurs, and gives rise to definite circular ulcers, which coalesce and ultimately involve a considerable area of the mucous membrane. These ulcers usually have their bases situated in the submucous coat, but occasionally they penetrate deeply into the muscular tunic, and may even lay bare the serous covering of the bowel. In severe cases the inner surface of the colon, sigmoid flexure and rectum may be completely honeycombed by the ulcerative process, the only remains of mucous membrane to be observed consisting of a network of indurated and discoloured tissue which serves to separate the various ulcers from one another. As a rule, the lower end of the large intestine suffers more severely than the upper portion, and in some cases the rectum is the only part of the bowel affected with ulceration.

MORBID HISTOLOGY.

The earliest description of the histological appearances presented by the stomach and intestine in cases of 'infantile atrophy' is to be found in the writings of Parrot,[1] who recognised the signs of chronic inflammation in many of the cases which he examined with the microscope. Since these observations were published, the subject has attracted comparatively little attention, and most writers, while they admit the presence of chronic gastro-intestinal catarrh, appear to attach but little importance to the organic changes induced in the digestive tissues by the inflammatory disease. Several years ago, being then unaware of Parrot's monograph on athrepsia, I commenced a systematic examination of the digestive organs of children dying from different forms of disease. Among these were 16 cases of infants who had succumbed to progressive emaciation, and in whom the post-mortem examination threw no light whatever upon the cause of the fatal symptoms. But in every instance the microscope demonstrated at once the presence of organic changes of an important nature in the alimentary tract, and it is chiefly from these cases that the few remarks which I have to make concerning the morbid histology of the disease have been compiled.

---

[1] *Op. cit.*

The Stomach.—The first sign of disease in the stomach consists of an infiltration of round cells into the interstitial connective tissue of the mucous membrane, similar to that already described in cases of acute gastric catarrh. The chief seat of the mischief is situated between the ducts of the gastric glands just beneath the superficial epithelium, the columnar cells of which are either detached or else converted into goblet cells. As the disease pro-

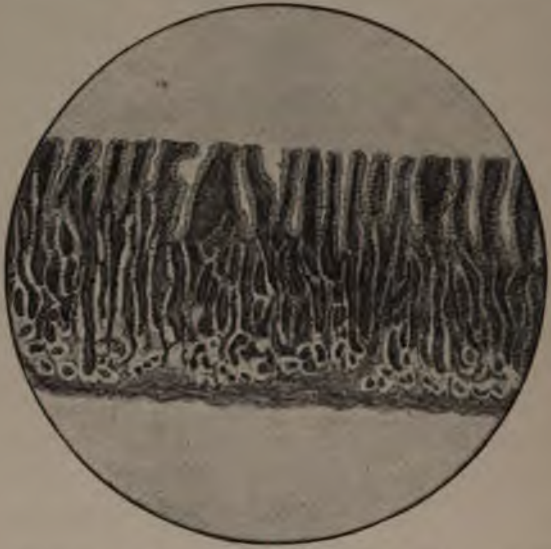

FIG. 4.—Section of a Normal Infant's Stomach. (× 80.)

gresses, the inflammatory process extends in the direction of the muscularis mucosæ, so that eventually the whole of the tissue between the glands becomes densely packed with deeply-staining nuclei, among which the original connective-tissue elements can often be discerned in a state of active proliferation.

The engorgement of the capillary vessels which ramify in the mucous membrane increases the pressure already exerted upon the tubules, so that the latter become displaced and their outlines obscured. The solitary lymphatic glands also undergo an increase in size owing to the proliferation of their cellular elements, and tend to encroach upon the free surface of the mucous membrane (Fig. 5).

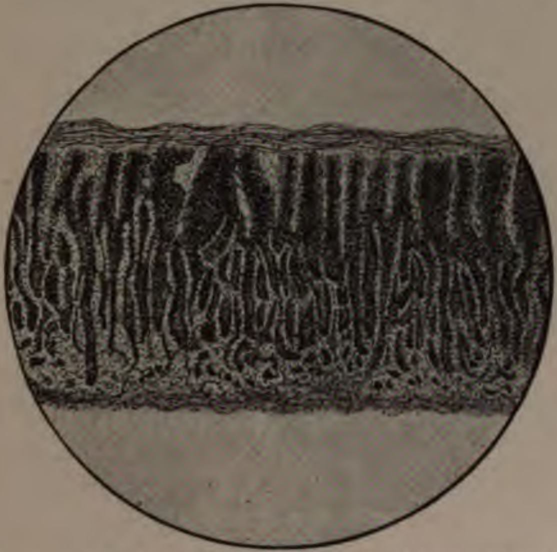

FIG. 5.—Section of the Stomach in an Early Case of Gastro-intestinal Catarrh, showing the Infiltration of Round Cells between the Glands of the Mucous Membrane (first stage of the disease). (× 80)

The second stage of the process is marked by the gradual organization of the inflammatory products. The superficial epithelium is now completely detached, and the surface of the section exhibits a jagged and uneven appearance owing to the irregular contraction of the newly-formed fibrous tissue. Instead of pursuing a course perpendicular to the surface, the ducts of the glands are seen to be twisted and distorted by the pressure exercised upon them, while their lumina are often choked with mucus, detached cells and epithelial débris. The tubular glands themselves are separated from one another by strands of fibrous tissue, the thickness of which varies at different spots. The basement membrane of the glands is thickened, and the secretory epithelium undergoes a series of changes as a result of the interference with its nutrition. In the cardiac two-thirds of the stomach, where the tubular glands are straight and comparatively short, the peptic cells usually proliferate at first, and entirely block the lumen of the gland. Afterwards they undergo fatty degeneration, so that eventually it is impossible to distinguish the outlines of the various cells amongst the mass of fatty and granular material which fills the greater portion of the tubule. In the pyloric region of the organ, on the other hand, where the gastric glands are of much greater length, and usually convoluted, the first effect of the contraction of the interstitial connective tissue is to constrict some portion of the tubule, and thus to impede the evacuation of its secretion. As the result of this, the lower end of the gland becomes dilated, while its epithelium loses its characteristic features and is gradually flattened against the basement membrane. In this manner a retention cyst is formed in the substance of the mucous membrane, which eventually becomes lined with a single layer of cubical epithelium. In certain sections, the solitary glands may be observed to have discharged their contents into the cavity of the stomach with the production of a follicular ulcer, while here and there a small hæmorrhage or minute erosion may be discerned upon the surface of the mucous membrane. These latter appearances are, however, merely accidental, and are devoid of any special significance.

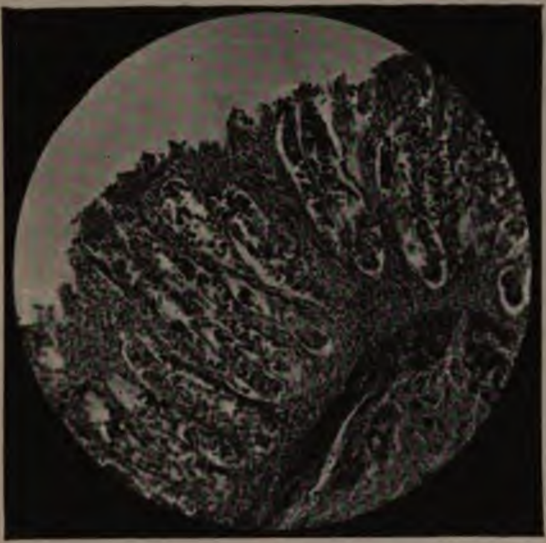

FIG. 6.—Photo-micrograph of the Stomach in a Case of Chronic Gastro-intestinal Catarrh, showing the Formation of Fibrous Tissue between the Gastric Glands (second stage of the disease). (× 80.)

At this period of the disease the submucous coat of the organ often participates in the inflammatory condition, and presents a general engorgement of its bloodvessels, with a considerable increase in the number of nuclei scattered through its tissue. The muscularis mucosæ is also infiltrated by small round cells, and in many places the contractile fibres show signs of compression by newly-formed fibrous elements. The muscular coat of the organ seldom presents any morbid phenomena beyond extreme engorgement of its vessels and an increase in the number of nuclei situated in its interstitial connective tissue.

The third and last stage of the catarrhal process is characterized by complete cirrhosis of the mucous membrane, with secondary changes in the other coats of the stomach. In many cases the surface of the section presents a peculiar papillary or villous appearance, which at first glance may cause the tissue to be mistaken for the small intestine. These spurious villi are due to the accumulation of the products of inflammation between the mouths of the ducts, and consist of round and spindle cells with capillary vessels of new formation. The gradual contraction of the fibrous tissue which has formed in the substance of the mucous membrane has given rise to atrophy of the gastric glands, so that the greater part of the section appears to be composed of fibrous elements, among which are scattered the remains of the tubules, the cells of which are usually in an advanced state of fatty degeneration. The cirrhotic tissue is extremely vascular, and when artificially injected exhibits numerous newly-formed vessels. It is probably on this account that the atrophic mucous membrane presents so slight a deviation from the normal when viewed with the naked eye. At this stage of the complaint the muscularis mucosæ is more or less completely destroyed, and the few strands of muscular fibres which remain appear to be embedded in a mass of fibrous tissue, and present signs of fatty degeneration. The submucous coat is much thickened and condensed by the organization of the inflammatory products effused into its connective tissue, while the arterioles which pass obliquely upwards to supply the mucous membrane exhibit sclerotic changes in their inner and middle tunics, and are not infrequently filled with thrombi of old formation. The muscular coat is often closely intersected by bands of fibrous tissue, and the contractile fibres appear granular or fatty. In very chronic cases the wall of the organ may be reduced to almost one-half of its normal thickness. Although the whole of the stomach is usually involved in the inflammatory process, the disease is found to vary in severity at different spots.

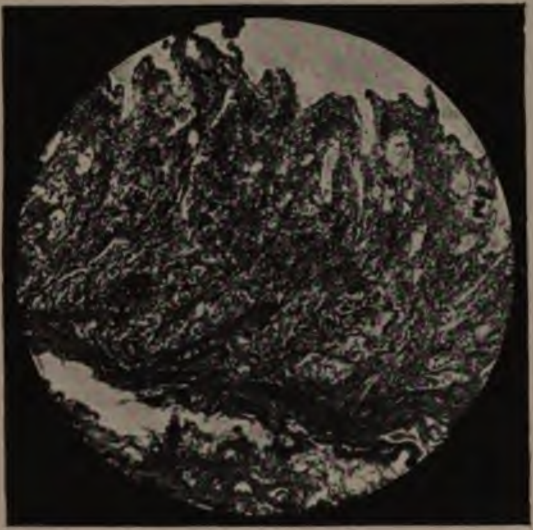

FIG. 7.—Photo-micrograph of the Stomach in a Case of Chronic Gastro-intestinal Catarrh, showing Cirrhosis of the Mucous Membrane, with Complete Atrophy of the Gastric Glands (third stage of the disease). (× 80.)

As a rule, its effects are most noticeable in the region of the lesser curvature, where the cirrhotic changes may give rise to an appearance of superficial scarring. In other parts of the organ the disease exhibits a more irregular distribution, so that it may frequently be observed that, while at one spot the mucous membrane is already in an advanced state of cirrhosis, at another in the immediate neighbourhood the inflammatory disease has only attained the second stage. These facts will be referred to later, when the question of recovery is discussed.

The Small Intestine.—The small intestine presents the same general appearances as have already been described in the case of the stomach. The vascular injection which accompanies the first stage of the disease is associated with an infiltration of small round cells into the interstitial connective tissue of the villi, and into that which separates the glands of Lieberkühn from one another. When organization of the exudation takes place, the contraction of the fibrous tissue obliterates the glandular structures, so that in severe cases the mucous membrane becomes more or less completely cirrhosed (Fig. 8).

At an early period in the disease the columnar epithelium which normally covers the surface of the bowel is detached, and many of the villi become adherent to one another as a result of the inflammation of their structure. Thus, in some sections, the surface of the mucous membrane presents a series of hoops or arches, owing to the adhesion of the villi at their free extremities, while in others the distinctive features of the tissue are obliterated through agglutination of the contiguous villi by their lateral margins. In many cases the epithelium of the tubular glands desquamates, and the ducts become blocked by masses of granular or fatty material; but in others the cells of Lieberkühn's glands preserve their normal appearance throughout. As soon as the contraction of the interstitial tissue exerts pressure upon the ducts of the glands, the tubules commence to dilate, and are eventually converted into small retention cysts, similar in structure to those already described in the case of the stomach.

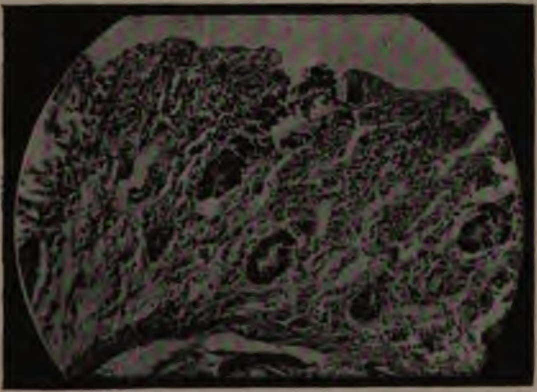

FIG. 8.—Photo-micrograph of the Small Intestine, in a case of chronic gastro-intestinal catarrh, showing Cirrhosis of the Mucous Membrane, with Atrophy of the Glands. (× 80.)

Brunner's glands, on the other hand, appear for the most part to escape the results of retention of their secretion. I have, however, frequently observed well-marked fatty degeneration of the epithelium of these convoluted tubules before the disease in the mucous membrane has advanced beyond the initial stage. At a later period of the complaint, when considerable induration of the mucous and submucous coats has taken place, the basement membrane of Brunner's glands, as well as the connective tissue between them, becomes remarkably thickened, and the secretory epithelium degenerates and is detached.

The Large Intestine.—In the large intestine the chief signs of disease are to be found in the transverse and descending portions of the colon, and in the rectum. The superficial epithelium usually persists for a considerable time, and its columnar cells may often be recognised, in a degenerated condition, upon the surface of the mucous membrane, when the latter is already in an advanced state of disease. As a rule, the catarrhal process is most pronounced in the centre of the mucous coat of the bowel, so that when fibrous tissue forms the tubular glands appear to be divided through the middle by a fibrous band. The epithelium lining the ducts is partially transformed into cells of the goblet type; while the rest becomes detached and forms fatty and granular plugs, which are gradually extruded from the mouths of the glands by the mechanical pressure of the inflammatory exudation (Fig. 9). When the products of inflammation undergo organization, the tubular glands become constricted about their centres, and their fundi are eventually converted into flask-shaped retention cysts. In the last stage of the disease the whole of the mucous and submucous coats of the bowel are completely cirrhosed. Occasionally the columnar cells lining an apparently healthy gland are observed to contain a quantity of golden-yellow pigment, as though they were in the act of excreting some product of blood disorganization. In very chronic cases large thin-walled vessels may often be seen coursing through the cirrhotic tissue, and forming sinuses of considerable size close to the free surface of the diseased bowel. This remarkable vascularity of the newly-formed fibrous tissue is probably the cause of the hæmorrhage which so often occurs at each act of defæcation.

Among the organic changes in other viscera which are apt to be associated with chronic gastro-intestinal catarrh, disease of the kidney is, perhaps, the most important. In his excellent monograph upon 'Athrepsia,' Parrot describes three morbid conditions of the renal organs which he had encountered in cases of infantile atrophy: fatty degeneration (steatose), thrombosis of the renal veins, and deposition of urate of sodium in the substance of the organ.

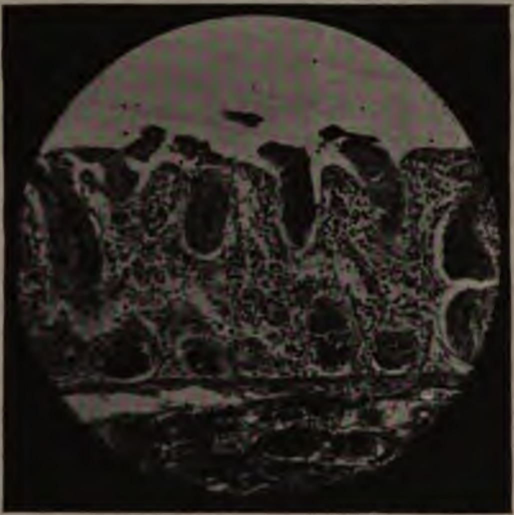

FIG. 9.—Photo-micrograph of the Large Intestine in a Case of Chronic Gastro-intestinal Catarrh, showing Partial Destruction of the Glands of the Mucous Membrane. (× 80.)

Fatty degeneration of the kidney is of comparatively rare occurrence. When it exists, the organ is slightly increased in size, and presents a pale yellow or mottled appearance, after the capsule has been removed. The cortex is increased in thickness, and at its periphery and near the apex of the pyramids yellow striæ may often be observed. Under the microscope the vessels of the Malpighian bodies are found to be engorged, while the walls of the glomeruli exhibit an excess of nuclei. The first sign of disease consists of an increased opacity of the protoplasm of the columnar epithelium, which gradually resolves itself into a large number of minute granules. These coalesce to form refractile droplets of some size, which stain black with osmic acid, and finally accumulate in such numbers as to completely block the tubules.

Chronic parenchymatous nephritis has been described by certain writers as a frequent complication of chronic catarrh of the gastro-intestinal tract in infancy, but in only one of the seventeen cases which I especially examined with reference to this point did the tubules show any signs of inflammation; while Holt only detected renal disease in one case among the series he investigated.

Thrombosis of the renal veins is usually confined to cases of a very chronic and severe nature. When it exists, the kidney is found to be enlarged and its capsule tense. Scattered over the surface of the organ are numerous small areas of a purple colour, which upon section are found to extend for some distance into the substance of the viscus. The apices of the pyramids are also deeply congested, and exhibit a blue or black coloration. The medium-sized branches of the renal ns are filled with grayish-white ante-mortem clot, e the larger veins usually contain coagula of recent ation. In rare instances the thrombosis may extend as far as the entrance of the renal veins into the inferior vena cava. Capillary hæmorrhages upon the surface or into the tissue of the kidney are often observed, and in a considerable proportion of the cases an apoplectic condition of the adrenals may also be detected (Parrot, Valleix, Mattei).

Uratic concretions in the tubules of the kidney are by no means uncommon. As a rule they take the form of yellowish-red masses, situated near the apices of the pyramids, which shade off into the substance of the organ in fan-shaped lines of a pale yellow colour. Occasionally the calyces, and even the mucous membrane of the pelvis, are found to be thickly powdered over with amorphous urates.

When examined with the microscope, the straight tubules are found to be blocked by opaque granular cylinders, which under a high power are seen to be composed of a vast number of minute spherical crystals. Considerable discussion has taken place concerning the exact chemical composition of these crystalline deposits, Virchow maintaining that they consist of urate of ammonium, while other authorities (Parrot, West) consider them to be either uric acid or urate of sodium. It is probable that in the majority of cases the sodic salt is the chief constituent of the deposit.

Legendre was the first to show that *fatty degeneration of the liver* is a frequent result of chronic diarrhœa in children. In this condition the organ is seldom notably enlarged, but presents an anæmic appearance, while its tissue is extremely soft and friable. Under the microscope the cells in the outer zone of each lobule are seen to contain a large number of fat globules. Occasionally I have observed the muscle of the *heart* to present similar features, the musculi papillares of the left ventricle being, as a rule, the first portions of the organ to suffer. This fatty degeneration of the heart is possibly the cause of the sudden and fatal syncope which sometimes occurs in cases of chronic gastro-intestinal catarrh, and may also be responsible for the mitral systolic bruit which occasionally develops during the later stages of the disease.[1]

In every case where death has occurred from gradual failure of the heart, *congestion* and *œdema* of the *lungs* is found at the necropsy. In most cases catarrh of the bronchial tubes also exists, associated with more or less extensive *lobular collapse;* while in nearly 16 per cent. of my fatal cases death was directly attributable to an attack of *broncho-pneumonia.*

Pulmonary tuberculosis is also frequently encountered at the post-mortem examination, and there can be little doubt that many infants who are supposed to die from simple bronchitis in reality succumb to this form of lung mischief.

Capillary *hæmorrhages* into the meninges or upon the surface of the *brain* are comparatively common, while in some instances sanguineous effusions of considerable size occur in the region of the medulla or in the substance of the cerebrum. Thus Parrot observed an effusion of blood in the substance of the brain in five out of thirty-four cases of intracranial hæmorrhage.

Thrombosis of the cerebral sinuses is occasionally the immediate cause of death. The anterior longitudinal sinus is the one which is most commonly affected, but sometimes the lateral sinuses or some of the smaller veins situated in the corpus striatum or upon the surface of the brain are affected in a similar manner.

---

[1] Author, *Brit. Med. Jour.*, May 23, 1891.

Finally, it may be mentioned that, in a large percentage of the cases of chronic gastro-intestinal catarrh, the *bronchial* and *mesenteric glands* are found after death to be enlarged and *caseous*, and are not infrequently affected with miliary tubercle.

SYMPTOMS.

The symptoms of chronic gastro-intestinal catarrh usually commence in an insidious manner, and often develop by almost imperceptible degrees from those which accompany the antecedent condition of dyspepsia. When the disease follows immediately upon an attack of acute catarrh of the digestive tract, the urgency of the vomiting and diarrhœa gradually abates, but the infant continues to reject its food at intervals, and to void three or four loose motions during the course of each day. These symptoms are accompanied by anæmia, fretfulness, and a steady loss of flesh, and as a rule, also, by an elevation of the temperature at night. Among the hundred cases of the disease to which allusion has been made, nine were stated to have commenced in an acute manner, and it is interesting to observe that in four of these the complaint ensued immediately upon an attack of measles. In the remaining ninety-one cases, the disease followed chronic indigestion, the transition from one complaint to the other being unattended by any phenomena of sufficient importance to attract the notice of the parents.

Following the teaching of Parrot, it has been the custom to divide the clinical course of the disease into three stages, the first of which is characterized by the prominence of the gastro-intestinal symptoms, the second by wasting of the tissues, and the third by certain cerebral phenomena indicative of exhaustion. Although this sequence of events may be observed in many chronic cases, in a very large number the different periods either run concurrently or even appear in reverse order. Thus, cases are frequently met where progressive emaciation and anæmia constitute the primary and sole important symptoms of the malady, notwithstanding the fact that both the stomach and intestine are found after death to be in an advanced state of disease. In other cases, again, loss of flesh or cerebral symptoms are present from the commencement, and continue throughout the whole course of the complaint. For these reasons I shall merely offer a general description of the disease and its complications, without endeavouring to draw any hard and fast line between its different stages.

Gastric and Intestinal Symptoms.

Symptoms indicative of a catarrhal state of the stomach or intestine are present in every case at one period or another during the course of the complaint. As a rule, a loose condition of the bowels or an abnormal appearance of the stools is the first symptom to attract attention. When the chronic disease follows immediately upon an acute attack, the stools usually number from four to seven a day, and consist of a dirty green or muddy-yellow liquid, alternating occasionally with others of a pasty consistence and paler tint; but after a time the tendency is for the motions to become quite diffluent, and to exhale an intolerable stench.

In the more insidious variety, on the other hand, the stools are passed less frequently, and exhibit remarkable variations from time to time in their naked-eye appearances. As a rule they are abnormally copious, and often seem to exceed in volume the total amount of nourishment consumed by the child. At one time the stools may consist entirely of a pale gray and putty-like material, in which a few lumps or streaks of green are visible; while at another they may be composed of a curdy or flaky substance tinged with green and mixed with a small quantity of fluid. At other times, again, the dejecta may closely resemble chopped spinach; or perhaps several ounces of a thin, opaque liquid like gruel may be evacuated from the bowel. In every case the motions possess a most offensive odour, which clings obstinately to the napkins despite repeated washing. With the progress of the disease the stools become diminished in quantity and more fluid in character, and although even at an advanced stage of the complaint they may still present a green and curdy appearance, they more usually consist of a brown and slimy material, containing traces of blood. Occasionally the infant habitually voids a jelly-like substance entirely composed of mucus, or every stool contains a large number of opaque pellets and shreds of the same material.

Hæmorrhage from the bowel is by no means infrequent in the later stages of the disease, each evacuation being found to contain streaks or clots of bright blood. When the act of defæcation is accompanied by tenesmus, a small quantity of liquid blood is often voided with each motion, but in many of these cases the source of the bleeding is to be found in a prolapse of the mucous membrane of the bowel. As a rule, the frequency of the diarrhœa varies inversely with the severity of the vomiting, and when the latter is a prominent symptom of the case, the bowels may be constipated rather than relaxed. Sometimes the diarrhœa presents lienteric features, each meal being immediately followed by the passage of a stool consisting mainly of undigested food. This form of intestinal flux appears to arise from an abnormally irritable state of the bowel, which causes it to be thrown into a violent contraction as soon as food is introduced into the stomach. At the commencement of the disease the stools are invariably acid to litmus-paper, and contain an excess of lactic and other organic acids; but as the complaint progresses the dejecta tend to become less acid, and in advanced cases they may be either neutral or even slightly alkaline in reaction.

Although *vomiting* is commonly supposed to accompany every case of chronic gastric catarrh, it is surprising how few infants with the disease suffer from *continued* emesis. Thus, only 44 per cent. of my cases exhibited this symptom in any marked degree, notwithstanding the fact that a chemical examination of the gastric contents usually indicated the existence of severe anatomical changes in the mucous membrane of the stomach.

In some cases, however, the infant vomits constantly during the whole course of the disease, the milk being rejected in a curdled state almost as soon as it is swallowed. Even in the absence of food the child may be subject to severe attacks of retching, which have for their object the expulsion from the stomach of small quantities of sticky mucus. When this condition continues unrelieved, the symptoms of emaciation and exhaustion make rapid progress, and the case often terminates fatally within a few weeks. In other instances vomiting is only of occasional occurrence, unless it be directly provoked by overloading the stomach with food.

All cases are liable to suffer from intercurrent attacks of subacute gastric catarrh, which usually persist for three or four days, and are accompanied by severe vomiting, and by rapid wasting of the tissues.

Pain in the abdomen is experienced in almost every case at one time or another during the course of the disease. It is most common at an early stage, before the diarrhœa has become fully established, and usually subsides to a great extent as soon as the motions assume a liquid character. The symptom is of a colicky nature, and arises from the presence of irritant materials or gas within the inflamed bowel. In cases of lienteric diarrhœa, sudden and severe griping in the umbilical or hypogastric region precedes, and perhaps accompanies, each action of the bowels. The infant expresses its sense of the abdominal discomfort by constant contraction of the muscles of the forehead and face, spasmodic flexion of the legs and thighs, and by frequent twistings and writhings of the body. In long-standing and neglected cases the flexor muscles of the lower limbs become permanently contracted, so that it is impossible to straighten the legs without the use of an anæsthetic.

General Symptoms.

After the lapse of a period of time, which varies with the severity of the digestive disorder, the child commences to lose flesh. At first it may merely be that the body-weight fails to exhibit its usual weekly increase. It soon becomes apparent, however, that an actual decline in weight is taking place, owing to wasting of the soft tissues. Loss of elasticity of the skin covering the deltoids, glutei and adductor muscles of the thighs is one of the earliest and most conspicuous signs of failure of nutrition, the superficial tissues becoming flabby, and capable of being pinched up between the fingers into loose folds. The subsequent absorption of the subcutaneous fat causes a peculiar wrinkling of the epidermis, so that after a short time it hangs loosely about the arms and thighs, and becomes arranged in a series of fine pleats along the inner margins of the buttocks.

Loss of flesh from the face tends to accentuate the lines which normally exist round the eyes and mouth, while the deep furrows produced by the incessant whining and crying of the infant give rise to that peculiar pinched, aged, and woebegone expression which is so characteristic of the disease. Next in order of frequency, the skin covering the abdomen and scapulæ exhibits the same flabby and wrinkled appearance ; indeed, so loose do the integuments become in these regions, that it often appears possible to grasp a handful of tissue, and to suspend the wasted infant, like a rabbit, by the skin of its back. Among the voluntary muscles, the adductors and extensors of the thighs, the gastrocnemei, deltoids and scaleni are the first to show signs of atrophy, and after a time present the appearance of thin cords when put upon the stretch. Finally, every muscle in the body, both voluntary and involuntary, participates in the general wasting, so that the long bones become denuded of flesh, and look like sticks covered with loose folds of skin. The bones themselves cease to increase in size at an early period of the complaint, and as soon as the soft tissues commence to waste, their compact tissue becomes gradually absorbed, and they consequently become fragile and prone to fracture. The bones of the head also suffer in a similar manner, more especially the upper and lower maxillæ, and this, combined with the wasting of the cheeks, makes the cranium appear of undue size, when compared with the face. In those cases, however, where vomiting and diarrhœa are very severe, the rapid loss of fluid from the body causes the anterior fontanelle to recede, and the bones of the vertex to overlap along their sutures, so that the whole head appears to diminish in size. Contrary to what is often observed during convalescence from other disorders of early life, children who recover from chronic gastro-intestinal catarrh usually remain stunted in stature for a considerable time, and, though strong and muscular, may continue unusually short, and often backward in mental development, for many years after the cure of the digestive disease. The actual loss of weight which occurs week by week varies according to the age of the child and the severity of the disease. Thus, in rapid and severe cases, as much as ten to sixteen ounces may be lost in a single week; but in the more usual and insidious form of the complaint the average does not exceed three to seven ounces in the same period of time. As the emaciation proceeds, the child begins to lose colour, and eventually becomes the subject of severe anæmia. In some cases the skin assumes a gray or clay-coloured tinge, while in others the face and hands acquire a faint yellow or bilious colour; but in all severe cases the anæmia eventually becomes extremely pronounced and progressive in character. The importance of this symptom will be further discussed when the subject of prognosis is considered.

The secretory functions of the *skin* become impaired at an early stage of the complaint, and the surface of the body appears dry and harsh to the touch. Cutaneous eruptions are common at all periods of the disease. Thus, strophulus existed in 28 per cent. of the cases which came under my observation. As a rule, the eruption principally affects the skin of the abdomen and face, but in severe cases the whole surface of the body may be closely studded with papules. In some cases the rash only makes its appearance in the evening, after the body has been washed, and is then attended with such intense irritation that the child is prevented from sleeping during the early part of the night. Eruptions of an eczematous nature are extremely common in advanced or neglected cases, and existed in 38 per cent. of my cases of the disease. As a rule, the scalp and posterior portions of the pinnæ are the parts most frequently attacked, but in severe instances the folds of the groins and axillæ, as well as the face, neck, buttocks, and thighs, may be affected in a similar manner. In very chronic cases, small boils, or subcutaneous abscesses containing curdy pus, are apt to make their appearance over the buttocks and backs of the thighs, and give rise to unhealthy-looking ulcers, which penetrate some distance into the subjacent tissues. The constant contact of the acid dejecta with the skin over the gluteal region causes it to become reddened and excoriated; while not infrequently superficial ulcerations occur round the anus and in its neighbourhood. In rare instances sloughing of the prepuce and scrotum, or gangrene of the vulva, is observed. In the later stages of the complaint the hands and feet often become puffy and œdematous, and even the skin of the legs and thighs may exhibit some degree of pitting upon pressure. This condition is usually ascribed to disease of the kidney, but in most cases it is due to a gradual failure of the circulation.

Chronic gastro-intestinal catarrh is usually accompanied by a remittent form of *pyrexia*, the internal temperature of the body rising to about 100° or 101° F. at night, and falling to 98° or 99° F. in the morning. In some cases short spells of fever alternate every few days with apyrexial periods, and in such it is not unusual to find after death that miliary tuberculosis has complicated the original complaint. Continuous fever, exceeding 102° F., generally indicates either a subacute attack of intestinal catarrh or some pulmonary or other serious complication. With the approach of death, the temperature of the body often rises rapidly, and may attain 106° F. either at or soon after the fatal event. At the commencement of the disease the *appetite* is well maintained, and the child often exhibits an insatiable desire for food; but as soon as the complaint has reached the chronic stage the appetite invariably declines, so that at the last it is almost impossible to persuade the child to swallow more than an occasional mouthful of nourishment.

Thirst is always present, more especially in those cases where diarrhœa or vomiting constitutes the principal symptom of the complaint. At first the *tongue* is somewhat redder than normal, and the dorsum presents a thick coating of grayish-brown fur; but after a time the organ tends to become pale and flabby, and is either quite clean or merely covered with a thin white fur. In the final stages of the complaint the mucous membrane of the tongue, as well as that of the buccal cavity and fauces, becomes the seat of parasitic inflammation, and small ulcerations make their appearance along the sides of the tongue and at the angles of the mouth. These morbid conditions of the mouth are of considerable importance, since they not only tend to destroy the appetite, but cause so much pain during the act of sucking or swallowing that the infant refuses its food and in consequence suffers from rapid emaciation.

The *urine* is always diminished in quantity, and when the diarrhœa is profuse only a few drachms may be passed in the course of the twenty-four hours. The fluid is usually turbid when freshly voided, and presents a copious sediment of uric acid and amorphous urates after standing for a short time. Micturition is sometimes accompanied by severe pain in the perinæum or glans, and in such cases it is not unusual to find crusts of crystalline material adherent to the orifice of the urethra or embedded under the prepuce. The total acidity of the urine tends to diminish during the course of the disease, but the daily quantity of the urea eliminated usually exceeds the normal until the body has undergone a considerable degree of emaciation. Many writers state that the disease is frequently associated with albuminuria and glycosuria. In order to test the accuracy of this statement, I had the urine drawn off in a large number of cases and carefully tested for these abnormal ingredients. In all, only 6 per cent. of the cases exhibited a trace of albumin, while sugar was never detected. These facts, taken in conjunction with the rarity with which parenchymatous nephritis is encountered after death, appear to me to indicate that renal inflammation is by no means so common a complication of chronic gastro-intestinal catarrh as is commonly believed.

The sediment of the urine usually consists of crystals of uric acid, mixed with a variable quantity of amorphous urates of sodium and potassium, but occasionally hedgehog crystals of ammonium urate or spherical masses of the sodium salt can also be detected. Epithelial casts are rarely encountered, but hyaline cylinders, either simple or filled with uratic salts, are by no means infrequent. Fatty casts I have never observed.

It is usually a matter of some difficulty to count the pulse and to estimate its volume in young infants affected with catarrhal disease of the digestive tract, while the heart-sounds are frequently obscured by rhonchi or by an abnormally harsh vesicular murmur. As a rule, in the early stages of the disease, the *pulse* varies between 90 and 110 per minute, according to the temperature of the body and the degree of exhaustion; but as the disease progresses the radial pulse becomes more and more feeble, and at the same time increased in frequency. It is a curious fact, however, that with the approach of death the action of the heart becomes slow, and often intermittent, so that perhaps not more than sixty beats can be counted in the minute when the organ is auscultated. The respiratory movements are increased in frequency during the early stages of the disease, even in the absence of any pulmonary complication; but as soon as the symptoms of exhaustion become pronounced, the breathing is usually slow and laboured, and may finally present the characters of the Cheyne-Stokes respiration.

The exhaustion of the nervous system which results from non-assimilation of the food, and the loss of fluid from the bowel, makes itself apparent in a profound alteration in the general appearance and behaviour of the child. The incessant screaming and whining which characterizes the initial stages of the complaint becomes replaced by apathy and depression, so that the child lies in its cot in a semi-somnolent state, and merely expresses its sense of discomfort by momentary contortions of the face or by an occasional moan. In this condition it may remain for many days, or even weeks, capable of swallowing food placed in its mouth, but otherwise exhibiting but little sign of life. Gradually the face becomes more and more pinched, the extremities cold and livid, the pupils dilated and sluggish in reaction, and finally death is ushered in so quietly that it is almost impossible to discern the moment when the spark of life is actually extinguished. In a large number of cases, however, the infant suffers from either local or general convulsions during the last stages of the disease, which are characterized by sudden rotation of the eyeballs, dilatation of the pupils, and tonic or clonic spasms of the muscles of the extremities. Sometimes these seizures are repeated at short intervals of time, the child remaining unconscious between the attacks, and finally succumbing to respiratory failure. In other instances the fits only occur once or twice, and apparently exert no deleterious influence upon the course of the disease.

Occasionally retraction of the head with strabismus appears to denote the presence of meningitis, but in such cases signs of inflammation in the brain can seldom be discovered after death.

COMPLICATIONS AND SEQUELÆ.

Among the numerous complications that are apt to occur during the course of chronic gastro-intestinal catarrh, those which affect the pulmonary organs are the most frequent and important. *Catarrh of the pharynx* is extremely common in cases where vomiting accompanies the gastric disorder, and not infrequently spreads to the mucous membrane of the larynx and trachea, and gives rise to the short, dry cough and hoarse cry so frequently observed. In more than half the entire number of my cases, examination of the chest revealed the signs of *bronchitis*, which in 29 per cent. was of sufficient severity to demand special treatment. *Broncho-pneumonia* existed in 13 per cent. of the cases, and affected one lung (usually the left) in 8 per cent., and both lungs in 5 per cent. In every instance where death ensued from gradual asthenia, the lungs showed signs of *hypostatic congestion* during the last few days of life, and in 3 per cent. of my cases moderate *effusion* into the right pleural cavity was recorded. Acute pleurisy, on the other hand, was never observed, except in association with pneumonia. In two cases, where the patients succumbed rapidly with the symptoms of cardiac failure associated with a high temperature, *purulent pericarditis* was found at the necropsy to have been the immediate cause of death ; but in neither instance was a friction sound detected during life.

The subjects of chronic gastro-intestinal catarrh are extremely susceptible to intercurrent attacks of *acute* or *sub-acute inflammation* of the *digestive tract.* This complication sometimes ensues as a result of some change of diet ; but during the summer months a large number of chronic cases are always attacked by the epidemic variety of intestinal catarrh. The occasional occurrence of *parenchymatous nephritis* has already been noticed ; but in my own practice I have only seen one case where œdema of the face and legs was associated with the presence of albumin and casts in the urine, and in this instance the infant eventually recovered. Other writers, however, have described a series of symptoms which attend the renal complication, among the most prominent of which are loss of elasticity of the skin over the abdomen, œdema of the extremities, vomiting, restlessness and convulsions, associated with a scanty and albuminous urine containing hyaline and epithelial casts.

Of the cerebral complications of the disease, *thrombosis of the venous sinuses* and *apoplexy* are the most important. Of the former I have only observed one case, although it is possible that many others may have escaped my notice, or have occurred after the patient's last visit to the hospital.

The profound malnutrition which accompanies chronic catarrh of the alimentary canal renders the subjects of the complaint unduly susceptible to the invasion of various *infectious diseases*, such as measles, scarlatina, and pertussis ; while in hospital practice, at any rate, acute *miliary tuberculosis* is responsible for a considerable proportion of the deaths. Even when a child recovers from the immediate effects of the disease, it is very apt to suffer from *rickets* during the period of convalescence. Thus, I find that no fewer than 27 per cent. of my cases which eventually recovered presented more or less pronounced signs of rickets before their final discharge from the hospital.

Chronic purulent discharges from the nose, ears or vagina are not infrequently encountered in these cases, and the enlarged glands which make their appearance in the neck or groin occasionally prove the starting-point of tuberculous adenitis.

There is, however, one result of chronic gastro-intestinal catarrh in infancy to which I would draw special attention, both on account of its practical importance, and also because it appears hitherto to have escaped attention. It is a well-known fact that infants who recover from chronic catarrh of their digestive organs usually do so in a slow and unsatisfactory manner, and not only continue very susceptible to attacks of gastric catarrh from exposure to cold or from some slight indiscretion in diet, but may remain for many years stunted in stature and deficient in strength and energy. But it is not so generally understood that the gastric disease contracted, and apparently cured, in infancy, is capable of exerting a deleterious influence upon the functions of digestion in adult life. Some years ago, when engaged upon a microscopical examination of the stomach in persons dying of different diseases, I frequently observed small patches of cirrhotic tissue in the mucous membrane near the lesser curvature. These signs of disease were obviously of old standing, and seldom appealed to the naked eye by indications more obvious than a slight pitting or puckering of surface. This subject was endowed with renewed interest when I afterwards had an opportunity of examining the digestive organs of children who had suffered during infancy from severe catarrh of the alimentary tract, and in whose stomachs well-marked patches of cirrhosis could be detected with the microscope. In these cases, also, the signs of disease were found chiefly, though not exclusively, near the lesser curvature of the organ. That this particular region of the stomach should so often present the relics of former mischief is hardly a matter for wonder, when it is remembered that the increase in the size of the organ which occurs after the second year of life mainly involves the cardiac and middle thirds of the viscus, and that consequently any permanent damage done to the mucous membrane in early infancy would always be most conspicuous in the pyloric, or least altered portion of the organ.

Now, there is a distinct clinical variety of dyspepsia which develops about the time that the growth of the body is completed, and proves most intractable to treatment. The fact that this complaint finds its chief victims among those who exhibit a sudden and remarkable rapidity of growth at a somewhat advanced age has led to the disorder being regarded as a result of 'outgrowing the strength.' But apart from the important distinctions which exist between the complaint in question and that which may reasonably be placed in this popular category, it is an interesting fact that in many of these cases evidence of the most convincing nature can be obtained of an attack of gastric or intestinal inflammation during the period of infancy. Thus, in several cases which have come under my immediate notice, the life-history of the patient, as obtained from the mother, was of the following kind: As an infant, the patient had seemed quite healthy until, owing to a sudden failure of the maternal milk or from some other cause which prevented suckling, recourse was had to artificial methods of feeding. As the result of this the child immediately became ill, lost flesh, and suffered from sickness and diarrhœa, and, in spite of repeated changes in the diet, became so weak and emaciated that the parents nearly abandoned hope of its recovery. Under medical care, however, it eventually improved, but remained for many years unduly small for its age, and suffered from obstinate constipation or from recurrent and severe 'bilious attacks.' After the age of fifteen the growth of the body was extremely rapid, and as soon as it was completed, symptoms of weak digestion, accompanied by atony of the colon, made their appearance, and had persisted ever since. Cases of this nature constitute a distinct class of habitual dyspeptics, and are encountered in every variety of medical practice. Unless carried off by some intercurrent disease like pneumonia or tuberculosis, the victims of this disorder remain for the greater period of their lives thin and anæmic, and not infrequently suffer from melancholia or hypochondriasis. The chain of evidence which connects cases of this nature with antecedent disease of the mucous membrane of the stomach and intestine is at present incomplete, inasmuch as it has hitherto been impossible to submit their stomachs to a microscopical examination; but there can be little doubt that chronic gastro-intestinal catarrh in infancy can leave its mark upon the digestive organs for many years, and exert an important and deleterious influence upon the functions of the stomach and intestine in later life.

PHYSICAL EXAMINATION.

As long as the catarrhal inflammation persists in a subacute form, the abdomen is found to be somewhat retracted, the superficial muscles rigid, while pressure with the hand gives rise to pain. In the chronic condition, on the other hand, the abdominal walls are usually flaccid, and the gaseous distension of the stomach and bowels which usually accompanies the disease may cause a perceptible separation of the recti muscles.

By careful examination, the existence of dilatation of the stomach can always be determined, and in many instances the lower border of the organ is found to extend below the level of the umbilicus. When a soft tube is passed into the stomach, and air pumped in by means of a hand-bellows, the viscus can be ballooned with the greatest ease, a fact which forcibly demonstrates the absence of tone in its muscular coat.

In those cases where the disease is not attended by vomiting, and where stagnation of the gastric contents habitually occurs, the characteristic splash can generally be obtained by appropriate manipulation. Occasionally the lower edge of the liver projects below the costal margin, while the left lobe of the organ can be detected in the left hypochondrium in close proximity to the spleen. When tenesmus, arising from follicular ulceration of the large intestine, is a prominent symptom of the case, the introduction of the finger into the rectum gives rise to pain, and often causes a reflex spasm of the anal muscles.

CHEMISTRY OF DIGESTION.

The morbid processes which occur in the mucous membrane of the stomach during the course of the disease give rise to important changes in the chemical composition of the gastric juice. In appearance, the contents of the organ vary considerably, but as a rule they are extremely viscid, owing to an excess of mucus. For this reason filtration is a matter of much difficulty, and it is often necessary to manipulate the material in its crude state. Its reaction to litmus-paper is usually acid; but in advanced cases of the disease the contents of the stomach may be neutral. In a certain number of cases (nine) I have been enabled to contrast the results of a chemical examination of the contents of the stomach made during life with the microscopic appearances presented by the organ after death, and these, when taken in conjunction with numerous other observations conducted at different periods of the complaint, permit of several general conclusions being arrived at.

In the *first stage* of the disease (Fig. 5), the contents of the stomach, when extracted one and a half hours after the test-meal, exhibit a large amount of mucus and much undigested curd. Leo[1] states that the total acidity in these cases is often higher than normal, owing to the presence of organic acids produced by fermentation; but I have never found it to exceed 0·135 per cent. HCl, while not infrequently it did not amount to more than 0·08 per cent. HCl.

One of the most important features is the invariable absence of free hydrochloric acid and the marked diminution in the quantity of the mineral acid combined with the proteid elements of the food. These facts are in strict accordance with our knowledge of the changes which occur in the gastric secretion from catarrh of the stomach in adult life. Lactic acid, as evidenced by a positive reaction with Ueffelmann's solution, is present in every case, and traces of butyric acid may frequently be detected. Under the microscope, pieces of curd, fat globules, epithelial cells, and numerous bacteria, can be observed.

In the *second stage* of the disease, where the gastric glands are beginning to suffer compression by the newly-formed fibrous tissue, the contents of the stomach still contain an excess of mucus, and often possess a pungent smell from the presence of butyric acid. Free hydrochloric acid is invariably absent, and the combined acid rarely exceeds 0·026 per cent., although, owing to the presence of secondary acids, the total acidity may reach 0·054 per cent. HCl.

In the *third* and *atrophic stage* of the complaint, the secretion of mucus usually fails, and the residue of a test-meal, even after a lengthy residence in the stomach, may possess hardly any appreciable degree of acidity. Pepsin may also be absent from the mixture, although both it and the rennet ferment can still be extracted from the mucous membrane by the introduction of dilute hydrochloric acid into the stomach.

---

[1] *Op. cit.*

The comparative slowness with which the stomach disposes of the food it receives affords important corroborative evidence of the enfeebled state of its muscular walls. Thus, in eight cases in which the symptoms of the complaint were supposed to have existed from two to four weeks, the organ was found to contain curdled milk from two and a half to two and three-quarter hours after the administration of two ounces of milk and water. In twenty-three instances, where the disease had existed from six to twelve weeks, the stomach was seldom found to be empty until after the lapse of three hours and a quarter; while in nine cases which exhibited a duration of more than three months, the organ often contained an appreciable amount of milk five hours after the meal.

EXAMINATION OF THE BLOOD.

It appears to be an established fact that at birth the blood is exceptionally rich both in corpuscles and colouring material. The exact number of the red corpuscles, as estimated by different observers, varies between 5,360,000 (Hayem) and 6,700,000 (Otto, Gundobin) per cubic millimeter, while about 12,000 to 15,000 white corpuscles are to be found in the same volume of blood. During the first few hours of life a slight increase in the number of corpuscles can usually be observed, but after the lapse of a day or two a steady decline sets in until the corpuscular richness approximates closely to that which obtains in adult life. The blood of a new-born infant is also peculiarly rich in hæmoglobin, but this likewise diminishes in quantity after the first week.

Quantitative estimation of the blood in infancy is beset with considerable difficulties, and the conclusions arrived at are often delusive, owing to the influence exerted upon the constituents of the blood by the state of the general health. Thus, it is found that a rise in the body temperature causes a rapid diminution in the number of the red corpuscles; while an attack of diarrhœa, probably by concentrating the fluid in the circulation, is accompanied by an apparent increase in the percentage amount both of corpuscles and hæmoglobin.

In thirty-one cases of chronic gastro-intestinal catarrh, regular estimations of the blood were made by means of the hæmacytometer and hæmoglobinometer of Gowers. The results of these investigations appear to show that at the commencement of the complaint a marked decrease in the percentage quantity of hæmoglobin can usually be observed. Thus, in fourteen cases where the symptoms of the complaint were stated to have lasted for less than three weeks, the colouring material varied between 65 per cent. and 78 per cent., while the corpuscular richness varied between 80 per cent. and 90 per cent. In every case the objective signs of anæmia were visible in the lips, conjunctivæ and skin. In one case of chronic marasmus, where microscopical examination eventually proved that the stomach and intestines were in a condition of moderate cirrhosis (Fig. 6), an examination of the blood made upon the day previous to death gave as results: corpuscles, 78 per cent. ; hæmoglobin, 66 per cent. In the last stage of the complaint, where the stomach is dilated, and the secretion of gastric juice reduced to a minimum, a steady diminution occurs, both in the number of the corpuscles and in the amount of hæmoglobin, so that in very chronic cases it is not unusual to find that the percentage of the former is less than 50, and of the latter less than 36. In such cases the anæmia is very conspicuous, and in many instances the loss of colour is so profound as to simulate the pernicious form of the disease in adult life. It is therefore not unreasonable to infer that a gradual destruction of the digestive and absorptive powers of the alimentary tract may constitute one of the numerous causes of severe anæmia.

PROGRESS AND TERMINATION.

The course pursued by the disease varies considerably in different cases. In out-patient practice it is usually observed that, when the complaint has once attained a certain degree of chronicity, it continues to make steady progress towards a fatal issue, owing to the adverse hygienic surroundings of the children of the poor, and the impossibility of providing the patients with those special forms of nourishment that are indispensable for the maintenance of health. When it is also remembered that in such cases medical advice is seldom sought until the disease has existed for many weeks, it is hardly surprising that chronic catarrh of the digestive tract in infancy is attended by such a long death-roll.

In the subacute form of the disease, where vomiting and diarrhœa are severe, death often occurs from asthenia or some intercurrent complaint between the fourth and the seventh weeks, or even earlier. But in the more chronic variety, where progressive emaciation and anæmia are the chief objective phenomena, the infant may linger for many months, and eventually succumb either to gradual exhaustion or to one of the numerous complications of the disease. Among the 100 cases which were selected for special examination, I find that no fewer than thirty-three died during the time that they were under treatment at the hospital. Even this high percentage probably underrates the actual mortality, since it was impossible to follow the ultimate progress of many of the remaining cases ; and I have a strong impression that if the truth could be ascertained, the death-rate from the primary disease or its sequelæ would be nearer 50 than 30 per cent.

Among the thirty-three cases which were known to have ended fatally, nine, or 27·2 per cent., died from asthenia, syncope, convulsions, or from some other cause directly connected with the primary complaint. In six cases (18·1 per cent.) death resulted from an attack of acute intestinal catarrh ; in five instances (15 per cent.) broncho-pneumonia was responsible for the fatal issue ; while in the remaining thirteen (39·7 per cent.) the infant contracted some infectious disorder, such as pertussis, measles, or scarlatina, which rapidly brought life to an end.

With regard to the actual duration of the disease in the fatal cases, such statistics as I can offer have only a limited value, owing to the habitual inaccuracy which characterizes all the statements made by hospital patients concerning the ailments of themselves and their children. As far as could be ascertained, however, in 61 per cent. of the cases the disease had lasted from one to two months; in 27 per cent. from two to four months; in 9 per cent. from four to six months; and in 3 per cent. for more than six months.

PROGNOSIS.

In the early stages of the disease, before the mucous membrane of the alimentary canal has suffered permanent injury, a cure can usually be effected by the strict observance of those elementary principles which should regulate the diet and hygiene of infancy. When, however, the stomach and intestines have become the seat of extensive cirrhosis, both digestion and absorption are dangerously impaired, and death will assuredly result, either from general malnutrition or from an attack of some intercurrent disease. The main point to determine, therefore, is whether the inflammatory mischief has produced irreparable damage to the digestive organs or not. Personally, I am in the habit of dividing cases of chronic gastro-intestinal catarrh into three classes, according to the length of time the disease has lasted, the severity of the general symptoms, and the functional state of the stomach, as determined by physical and chemical examination.

The *first* class includes all cases in which the complaint has commenced in an *insidious* manner, and has not lasted for more than two or three weeks. In such the main symptoms consist of a gradual loss of flesh, abdominal pain, flatulence, and loss of appetite. Vomiting only occurs at intervals or after a particularly copious meal, while the stools present a pasty appearance, alternating occasionally with others of a more liquid character. Bronchial catarrh, if it exists at all, only affects the larger tubes, and the blood contains more than 80 per cent. of red corpuscles, and 75 per cent. of hæmoglobin. Examination of the stomach after a test-meal shows that its motor activity is so far preserved that the organ is empty within two and a half hours; while the curdled milk which can be withdrawn at the end of an hour and a half presents signs of partial digestion. Although free hydrochloric acid is absent, the contents of the organ are very acid, and on examination it can be shown that the combined acid in the mixture is not appreciably diminished, while the total acidity does not fall much short of 0·1 per cent. HCl.

Cases which present these general features are extremely common, and constitute the bulk of the cases where progressive loss of flesh is the chief symptom of the disease. The prognosis is good, since the infant, if provided with diluted and sterilized cow's milk and cream, commences to improve at once, and eventually makes a perfect recovery.

In the *second* class, the disease has existed for many weeks, and has given rise to organic changes in the digestive organs. Vomiting is often observed and the motions are frequent, watery, and extremely offensive. Anæmia is present to a marked degree, and examination of the blood shows that its corpuscular richness is less than 80 per cent., while the percentage of hæmoglobin varies between 60 and 70. The stomach is invariably dilated, and contains food for three hours or more after the administration of a test-meal. The gastric contents exhibit an excess of mucus which prevents filtration; free hydrochloric acid is absent, and the quantity of the combined acid much diminished. The peptic and rennet ferments are still present, but the mixture after acidification digests slowly and imperfectly. Bronchitis is usually present, and not infrequently the mouth is affected with parasitic stomatitis, while the anus and thighs are excoriated by the alvine discharges. The body of the child is much emaciated, and the temperature elevated a degree or two each evening.

In these cases the prognosis, both immediate and remote, must be extremely guarded, for, in addition to the danger arising from the original disease, death is prone to occur at any time from pulmonary or other complications. When recovery sets in, the first sign of good omen is usually a cessation of the peevish crying, and a greater degree of restfulness at night. This is followed by increased appetite, diminished thirst, and a fall of the temperature. The body also ceases to lose weight, though it may be several weeks before the child can positively be said to be gaining weight. When this does take place, the face and neck are usually the first to exhibit an increase in the amount of subcutaneous fat. I have occasionally observed a rapid increase in the percentage of hæmoglobin to precede all other signs of returning health; while a marked improvement in the motor and secretory functions of the stomach may be regarded as certain evidence of ultimate recovery.

In the *third* class must be included those numerous cases where the protracted nature of the complaint has reduced the infant to a state of extreme emaciation and anæmia. Although the appetite may be ravenous, and the symptoms of disordered digestion but little apparent, the child continues to go from bad to worse, despite every care. Examination of the stomach shows it to be considerably dilated, and in the habit of retaining food for several hours; while its contents, when extracted an hour and a half after a test-meal, contain hardly any mucus, and are almost devoid of hydrochloric acid and pepsin. In very chronic cases, even the secretion of rennet is suppressed. The skin and conjunctivæ appear bloodless, and if any blood can be extracted by pricking the finger, it presents a pale-pink and watery appearance, and is found to contain less than 50 per cent. of corpuscles and hæmoglobin.

In such cases the prognosis is hopeless, for the atrophic condition of the mucous membrane of the alimentary tract precludes any possibility of the digestive organs regaining that degree of functional activity which is necessary for the continuance of life.

DIAGNOSIS.

The diagnosis of chronic gastro-intestinal catarrh seldom presents any material difficulty. It is true that loss of flesh and anæmia may accompany such diseases as congenital syphilis, rickets and tuberculosis, but in these cases some other symptoms of the complaint are invariably present and aid in its detection. Thus, in congenital syphilis, there is usually a history of snuffles, the nose is flattened, and scabs or sores may be observed about the nostrils or angles of the mouth. The skin of the body is dry and wrinkled, and often covered with a coppery eruption. Mucous tubercles can sometimes be detected in the mouth or round the anus, while the buttocks, hands and feet may present the red and polished appearance so characteristic of the disease. In the inflammatory disease, on the other hand, the skin has an earthy tint, and if any eruption is present, it is of a strophulous, and not of a specific, nature. The excoriated condition of the skin round the anus is also quite different from the appearance presented by the parts in cases of syphilis.

In acute general tuberculosis, the most noticeable symptoms arise from the implication of the pulmonary organs. The child coughs incessantly, and examination of the chest reveals either generalized bronchitis or patchy consolidation. The fever which accompanies the disease is of a remittent character, and much more severe than in cases of catarrh of the alimentary tract. The bowels are more often confined than loose in their action, and copious perspirations occur at night time. As the disease progresses, the legs and ankles become œdematous, and in many cases the meninges or peritoneum are invaded by tubercle.

When the wasting of the body arises from rickets, the changes in the bones of the skull or of the extremities, delayed dentition, the tumid belly, and other signs of the disease, are a sufficient guide to the nature of the complaint.

There yet remains one common cause of wasting of the body in infancy which unhappily demands more than a passing remark. I mean starvation pure and simple. In some cases this arises from sheer ignorance, and the parents are genuinely surprised when they are told that to feed an infant a few weeks old upon oatmeal, flour, vegetables, or meat, in lieu of milk, is tantamount to withholding food from it altogether. There are, however, other cases where, either from cruel neglect or criminal intent, the child habitually receives less than the minimum quantity of food necessary to support life, and consequently wastes and dies.

That the inanition is the result of starvation, and not of disease, is easily proved by the use of the gastric tube; for while in some cases it can be shown that the stomach is empty within an abnormally short time of the administration of the supposititious meal, in others the introduction of milk into the stomach easily demonstrates that there exists no lack of secretory power on the part of the digestive organs.

TREATMENT.

General.—Whenever the gastric or intestinal symptoms are severe, the child should be confined to its cot, and the air of the bedroom maintained at a temperature of about 65° F. In less urgent cases, the infant may be wheeled out into the fresh air once or twice a day, if the weather permits. A warm bath may be given night and morning, care being taken to avoid any exposure to cold. In every case the child should be warmly but not too heavily clad, and it is wise to keep a flannel bandage, or some carded wool covered with flannelette, constantly applied round the abdomen.

If the nates show any signs of redness or excoriation, they should be carefully cleansed once or twice a day with thick oatmeal and water, and afterwards dusted over with a powder composed of equal parts of oxide of zinc and starch, or, if necessary, dressed with zinc or boracic ointment. The napkins must be changed at regular intervals, and, along with any other soiled linen, should be thoroughly disinfected before being washed. If phimosis is present, an attempt must be made to dilate the orifice of the prepuce in order to promote the complete and regular evacuation of the contents of the bladder. Symptoms of collapse require to be combated by the application of warmth to the extremities, and by the administration of suitable stimulants; while thrush, and the various other complications of the disease, must be treated in the ordinary manner. As soon as convalescence is well advanced, much good will usually accrue from a few weeks' residence in the country or by the seaside.

When the stomach is much dilated and its contents exhibit an excess of mucus, it is wise to cleanse the organ before active treatment is commenced. For this purpose a soft catheter of moderate diameter should be employed, and warm water introduced into it through a glass funnel under atmospheric pressure. Occasionally, however, some difficulty is experienced in the evacuation of the contents of the stomach by the simple process of siphonage, and it is necessary to replace the funnel by a glass syringe, and to empty the organ by means of suction. For the purposes of lavage, pure water at the temperature of the body is to be preferred to anything else, the fluid being introduced slowly, and in quantities not exceeding two ounces at a time. The addition of a few grains of carbonate of sodium to each douche is of value in those cases where the vomit contains an excess of tenacious mucus. Personally I am opposed to the use of such antiseptics as resorcine, boracic acid, and naphthalin for the purposes of lavage, since absorption readily takes place from the stomach, and serious symptoms have been known to follow the use of even the least poisonous of these remedies. In most cases, it is sufficient to perform lavage once or twice a week, but in very severe and obstinate forms of the disease it may be necessary to cleanse the organ every morning.

The soft tube and funnel are also of considerable use in the performance of forcible feeding (Gavage). It is well known that many infants who reject everything they swallow will retain in their stomachs any food which has been forcibly injected into the organ. This fact is made use of in cases where persistent vomiting precludes the administration of nutriment by the mouth ; and it is often found that after forcible feeding has been employed for a short time, the tendency to emesis disappears. This method is also of value in the later stages of the complaint, when an absence of appetite prevents the child from taking a sufficient supply of nourishment.

Dietetic.—Since the majority of cases of chronic gastro-intestinal catarrh arise from improper feeding, the selection of a suitable dietary is a matter of the greatest moment. In breast-fed children the state of the mother's health and the condition of her milk must be made the subject of careful examination. Should she be found to be suffering from some grave constitutional or organic disease, or if the infant exhibits a marked distaste for the breast, and suffers from sickness or purging after each meal, it is advisable to prohibit nursing and to procure a wet-nurse instead. If, however, the child takes the breast with avidity, but appears dissatisfied at the end of the meal, it is probable that the symptoms of inanition result from the innutritious quality of the mammary secretion. In these cases the breast must be supplemented by cow's milk, or by some other form of artificial food.

In the case of hand-fed children, all varieties of milk and farinaceous foods must be suspended until the alimentary canal has been cleansed from the masses of curd and fermenting material with which it has been overburdened. The subsequent choice of a dietary must depend upon the severity of the digestive disease. When vomiting or diarrhœa constitute the prominent features of the case, fresh milk almost invariably disagrees, owing to its tendency to undergo fermentation in the inflamed stomach. It is necessary, therefore, to maintain the strength of the infant by the judicious employment of the various animal broths. For this purpose veal, mutton, or chicken broth may be given every hour in doses of a tablespoonful or more; or raw meat juice, Valentine's meat juice, or clear ox-tail soup may be used. After the lapse of twenty-four hours, equal parts of barley-water and whey may be tried, and if this agrees, a little cream may be added to the mixture. Should the irritability of the stomach continue, a few drops of brandy or champagne, or white wine whey, may be added to each meal. Should the vomiting still continue, lavage followed by forcible feeding must be employed. As the child continues to improve, the diet may be further strengthened by the addition of Mellin's food, bread jelly, cream mixture, or even the yolk of an egg beaten up with barley-water and brandy.

As soon as the gastric and intestinal symptoms have disease, especially when the children have been nourished entirely upon preserved milk and farinaceous foods, it is by no means uncommon to observe a tendency to swelling and ulceration of the gums. This condition is always accompanied by severe anæmia, and seems to be of scorbutic origin. In such cases the administration of a small quantity of orange or lemon juice every day is followed by a rapid improvement both in the general health and also in the state of the digestive organs. After the age of twelve months a little fresh vegetable should be included in the dietary, well-boiled onion, celery, asparagus, potato, or vegetable marrow being the most suitable.[1]

But, despite every effort, there will always be found a certain number of cases which are unable to take liquid nourishment without suffering from diarrhœa. In such the use of the raw meat pulp is often attended by very satisfactory results. The pulp must be prepared in a careful manner (R. 13), and given at first in teaspoonful doses at intervals of two or three hours. As a rule, children soon acquire a liking for the raw meat, but should any serious aversion be shown to it, the pulp may be mixed with a little sweet gravy or made into a jelly. As soon as tolerance has been established, the dose of the pulp should be cautiously increased, until the child can take half a pound of meat in the course of the twenty-four hours. At first the stools are apt to be somewhat increased in number, and are accompanied by a horrible odour of decomposition; but in the course of time, and especially if pepsine along with an antiseptic is administered after each meal, the motions assume a more natural appearance. The only danger attendant upon the use of uncooked meat lies in the possible introduction of some intestinal parasite; but careful selection and examination of the meat is usually sufficient to prevent this accident. It is a great mistake to discard the meat pulp as soon as the infant shows signs of improvement, for it too often happens that the premature use of milk or starchy foods is followed at once by a serious relapse.

---

[1] Henoch speaks of the value of a compot made from dried whortleberries in cases of obstinate diarrhœa; while in certain country districts acorn-tea is extensively used for a similar purpose.

The use of a diffusible stimulant is indicated in all cases which exhibit a tendency to exhaustion or heart failure. As a rule, the white wine whey (R. 15), when given in dessertspoonful doses, meets all requirements, but under exceptional conditions recourse must be had to good pale brandy, whisky, burgundy, or champagne. The brandy can be advantageously combined with egg, as in the brandy mixture of the Pharmacopœia.

Both koumiss and kéfir are spoken favourably of by certain writers, but as a rule their effects are disappointing.

Medicinal.—The principles which should regulate the medicinal treatment of the chronic disease are essentially the same as those adopted in the acute form of the complaint. If the diarrhœa is not urgent, and the stools contain lumps of undigested food which are passed with pain and straining, it is advisable to administer a purge, in order to rid the bowel of its irritant contents. For this purpose castor-oil, calomel, or rhubarb and soda may be employed, the first-named being usually the most satisfactory. Even in cases where the motions are liquid and passed with a moderate degree of frequency, small doses of the castor-oil mixture (F. 1), with perchloride of mercury, or calomel (one-eighth to a quarter of a grain), or gray powder (quarter to half a grain), given every three or four hours, are attended by beneficial results. After these measures have been persevered with for a day or two, active antiseptic treatment should be commenced. The generally accepted treatment of chronic diarrhœa consists in the administration of various astringent drugs which are supposed to exert a local influence upon the mucous membrane of the bowel, and to prevent the escape of fluid from its vessels. Accordingly, text-books are usually full of prescriptions containing such substances as sulphuric and nitric acids, hæmatoxylin, chalk, rhatany, catechu, tannic or gallic acid, alum, sulphate of copper, acetate of lead, etc. Some of these drugs possess an undoubted value when applied directly to the mucous membrane in the form of rectal injections; but their routine administration in relatively minute doses by the mouth is both unscientific in theory and valueless in practice. The inutility of the ordinary 'diarrhœa mixtures' in the chronic complaint of infancy must be patent to everyone who is in the habit of dealing with such cases; and from my own experience I can affirm that I have never seen a case cured by astringents in which a careful trial of antiseptics and sedatives had previously failed. On the other hand, it is a common experience to meet with cases where the substitution of rational methods of treatment for the empirical use of astringent drugs is followed at once by immediate improvement.

Of the various antiseptics, resorcine is, without doubt, one of the most useful, owing to its ready solubility, cheapness, sweet taste, and non-poisonous character. It must, however, be given frequently and in full doses (three to five grains), and may be advantageously combined with subnitrate of bismuth (F. 3). As a rule, the intestinal flux begins to abate after the eighth dose, and the continued administration of the drug is not infrequently followed by obstinate constipation. In other cases, carbolic acid, creasote, iodine, or perchloride of mercury may be tried (F. 3). When vomiting is severe, the salicylate of bismuth (three to five grains), either alone or combined with a minute dose of calomel, is attended by remarkably good results; or the salicylate of strontium (one to three grains) may prove successful when everything else has failed. Benzol-naphthol is of great service when the larger bowel is the chief seat of the disease, but it must be given in full doses (five to eight grains, every four hours), in order to produce a good effect.

It seldom happens that the diarrhœa arising from simple catarrh of the intestine cannot be controlled by one or other of these drugs. In very chronic cases, however, the symptom more often depends upon secondary follicular ulceration of the large bowel than upon simple catarrh of the smaller gut, and in these cases sedative remedies are to be preferred to antiseptics. In such, a small dose of Dover's powder combined with carbonate of bismuth and chalk answers extremely well, or the tincture of opium may be used along with one of the mineral acids (F. 2). This latter prescription is particularly useful in those cases where the diarrhœa is lienteric in character, or where the stools are habitually green in colour. Ipecacuanha is also a valuable remedy in these cases, and may be given either in the form of the powder or the wine; but it must always be combined with some aromatic or stimulant preparation, in order to counteract its depressant effects. In some instances it is most efficacious when minim doses of the wine are given every hour; while in others minute doses of the powder (one-sixteenth to one-eighth) combined with Dover's powder and chalk or bismuth, appear to answer best (F. 2). When severe vomiting follows each effort at defæcation, antimonial wine in minim doses every hour is often attended by success. In the most severe cases, where tenesmus is associated with the passage of blood and slime, and with prolapse of the rectum, an enema of opium and starch containing a small dose of ipecacuanha given twice a day is of great value; while in obstinate cases daily irrigation of the large bowel may be undertaken.

For the due performance of this operation, the patient should lie upon his right side, with the buttocks raised, and the fluid should be allowed to flow into the bowel at the atmospheric pressure. Simple warm water (temp. 65° F.) or a dilute solution of common salt ($\frac{1}{2}$ per cent.) act as sedatives to the inflamed mucous membrane, and aid in the removal of the acid mucus or other irritant material which may be present. Sometimes weak solutions of acetate of lead ($\frac{1}{2}$ per cent.), nitrate of silver (one grain per ounce), alum (one grain per ounce), or salicylate of sodium (five grains per ounce), may be employed with advantage.

As soon as the immediate symptoms of the complaint have completely subsided, the exhibition of tonic remedies is indicated. The bland preparations of iron are particularly useful, though occasionally the perchloride is also beneficial. Nux vomica and arsenic are valuable remedies at this stage of the complaint, and may be advantageously combined with some bitter infusion. In cases where the digestion continues feeble during the period of convalescence, nothing is so valuable as the glycerine of pepsin administered immediately after the meals. Cod-liver-oil should not be given as long as any tendency to diarrhœa exists.

CHAPTER VI.

ACUTE GASTRIC CATARRH IN CHILDREN

(EMBARRAS GASTRIQUE—ACUTE INDIGESTION—BILIOUSNESS—STATUS GASTRICUS — SABURRAL CONDITION — GASTRIC FEVER — TOXIC GASTRITIS).

ETIOLOGY.

PREDISPOSITION to acute catarrh of the stomach may be hereditary or acquired. It is by no means unusual to find that several members of a family will develop the symptoms of gastric catarrh when exposed to influences which prove quite harmless to individuals of a less susceptible nature. This peculiar disposition can often be traced back in the family for several generations, and may possibly be the explanation of many of those idiosyncrasies of digestion which so many persons appear to inherit from their forefathers.[1] It is also a noteworthy fact that, while the members of one family will contract the disease owing to a change in the temperature or degree of humidity of the atmosphere, those of another will only develop the symptoms after partaking of some special form of food; others again, are unaffected by both of these conditions, but will immediately suffer whenever an epidemic of an infectious disorder, like scarlatina, typhoid, or 'summer diarrhœa' breaks out in their neighbourhood. Family predisposition was a conspicuous feature in nearly 17 per cent. of all the cases of acute gastric catarrh which have come under my notice at the Evelina Hospital and elsewhere, and I am consequently inclined to invest it with a much greater degree of importance than it has hitherto been accorded.

---

[1] Examples of this are to found in the severe gastric symptoms produced in certain persons by the use of butter, fat, pork, game, mackerel, crab, oatmeal, cucumbers, strawberries, apples, mushrooms, pickles, chestnuts, almonds, cubebs, and honey.

Predisposition to catarrhal affections of the stomach may be *acquired* in many ways. One attack of the disease always invites another, so that a child who has once suffered from the complaint remains for a long time unduly susceptible to those causes which usually excite it. Certain constitutional affections, like marasmus, scrofula, syphilis, and rickets, also favour the inception of the malady; while children who are the subjects of renal, cardiac, or pulmonary mischief are apt to develop the complaint upon the slightest provocation. Finally, it may be noticed that the convalescent period of measles, scarlatina, whooping-cough, and other specific infectious diseases, is always fraught with danger from a similar cause.

For the purpose of etiological description, acute catarrh of the stomach may be divided into two varieties—primary and secondary—according as the disease develops in an otherwise healthy subject, or appears as a complication of some other and more serious condition.

Acute Primary Catarrh. — Among the exciting causes of this affection, exposure to cold and wet is usually considered of much importance. The greatest danger from this source arises during the spring and autumn months, when sudden variations are apt to occur in the temperature of the air, while the atmosphere becomes damp and foggy after sunset.[1] Gastric catarrh from this cause is often directly courted by the observance of that curious domestic custom which ordains that children at the most delicate period of their lives should parade about in all weathers with bare arms and legs, while such clothing as they possess is piled upon the chest. That this wholesale exposure to cold is a fertile source of the disease in early life is easily proved by the fact that the recurrent tendency to the complaint can often be abolished at once by clothing the child in a rational manner. At certain periods of the year catarrh of the stomach is apt to appear in an epidemic form, and is not infrequently associated with a similar affection of the nasal and bronchial mucous membranes. It is probable, however, that in this type of the disease, the gastric disorder is merely one of the results of a general infection of the system.

Direct irritation of the stomach by abnormal products of digestion is an extremely common cause of gastric catarrh in early life. Under normal conditions the gastric juice possesses powerful antiseptic properties, most of the micro-organisms which find an entrance with the food being either inhibited in their growth by the mineral acid, or actually digested and destroyed by the gastric ferment. But if from any cause the secretion of hydrochloric acid is diminished, the bacteria flourish and multiply in the stomach, and by their action upon the food give rise to the formation of certain organic acids, which in their turn exert an irritant influence upon the mucous membrane of the digestive tract. There is also another way in which the ingestion of food may give rise to inflammation of the stomach. In certain cases, the rapidity of the onset and the severity of the symptoms point to the introduction of already-formed chemical poisons. These toxic principles are usually of an organic nature, and owe their origin to decomposition of the food through the agency of certain bacteria. Examples of this nature are to be found in the violent gastro-enteritis which sometimes follows the ingestion of high game, putrid fish, or tinned meats. Occasionally the complaint arises from contamination of the food during the process of cooking by such mineral poisons as copper, zinc, lead, and arsenic. It is also possible that acute catarrh of the stomach may sometimes ensue from the use of foods which are either too cold or too hot. Thus, I have more than once seen cases where the first symptoms of the disease manifested themselves shortly after a child had sucked a large quantity of block ice, or had eaten several ices in rapid succession, and in which there was no reason to suppose that the ice itself contained any elements of a poisonous character. Conversely, the ingestion of abnormally hot substances may also induce an inflammatory state of the gastric mucous membrane, the offending materials appearing literally to scald the fundus of the stomach. In this connection it is interesting to note that Decker[1] was able to produce severe inflammation, and even ulceration, of the stomach by feeding dogs through a tube with food heated to 120° F.

---

[1] Willigk (*Prager Vierteljahrsch.*, li., p. 28) gives for 327 cases of gastric catarrh the following relative proportions between the number of cases occurring at different seasons: Spring, 6·2; summer, 3·4; autumn, 2·9; winter, 2·5.

---

[1] *Brit. Med. Journ.*, 1887.

There yet remains to be mentioned one variety of gastric catarrh which possesses particular interest, inasmuch as it occurs during epidemics of enteric fever, which disease in its general symptoms it closely resembles. Outbreaks of this nature have been described by Chantemesse,[1] Grimshaw,[2] and others; while the first-named has shown that the distribution of the water of the Seine among the inhabitants of Paris is regularly followed by an increase in the number of cases both of typhoid fever and of acute gastric catarrh. Other epidemics of gastro-enteritis have been traced to the use of impure water or milk; and in one instance Gaffky[3] believes that he was able to demonstrate as the exciting cause of the disease an extremely virulent form of the *Bacterium coli commune.*

Acute gastric catarrh of *secondary* origin is much less common in children than in adults. It chiefly occurs during the early stages of certain specific fevers, like scarlatina, measles, small-pox, erysipelas, influenza, and pertussis; or as the result of chronic disease of the heart, lungs, liver, or kidneys. It is also apt to occur when the mucous membrane is already in a condition of chronic inflammation. Occasionally the disease is accompanied by the formation of a false membrane, as in that variety which sometimes complicates diphtheria.

---

[1] *Semaine Médicale*, November 13, 1889.

[2] *Dublin Journ. Med. Sci.*, 1879, p. 269.

[3] *Deut. Med. Woch.*, 1892, s. 297.

MORBID ANATOMY.

The macroscopic appearances of acute gastric catarrh are extremely variable. As a rule, the stomach is found after death to be moderately distended with gas, and to contain a few ounces of bile-stained fluid. The inner surface of the organ is usually covered with a layer of mucus, which is especially thick and tenacious in the region of the pylorus. When this has been removed, the subjacent tissue appears to be soft, swollen, and opaque. In mild cases the mucous membrane exhibits a dead-white colour, and even in severe instances of parenchymatous inflammation there may be a complete absence of hyperæmia. In that variety of gastritis which arises from irritant poisoning, the mucous membrane of the stomach usually presents a considerable degree of superficial injection ; but even in these cases the colour of the tissue varies with the length of time which has elapsed between the death of the patient and the necropsy. As a rule, the signs of hyperæmia are limited to the summits of the rugæ in the fundus of the organ, but occasionally the whole of the inner surface of the stomach presents a swollen, glazed, and vivid red appearance. In severe cases capillary hæmorrhages are seldom absent, and sometimes the greater portion of the mucous membrane on the posterior surface and in the region of the lesser curvature is studded with punctiform extravasations of blood. Occasionally the area occupied by a single hæmorrhage may be as large as a pea, or even a threepenny-piece, but actual erosion of the mucous membrane is seldom encountered owing to the absence of an active gastric juice.

Most of our conceptions concerning the morbid appearances of the stomach in cases of acute catarrh have been derived from the observations of Beaumont[1] upon Alexis St. Martin, which cannot be expressed more vividly than in that author's own words: 'There are sometimes found in the internal coat of the stomach eruptions or deep red pimples, not numerous, but distributed here and there upon the villous membrane, rising above the surface of the mucous coat. These are at first sharp-pointed and red, but frequently become filled with white purulent matter. At other times irregular, circumscribed red patches, varying in size from half an inch to an inch and a half in circumference, are found on the internal coat. These appear to be the effect of congestion of the minute bloodvessels of the stomach. There are also seen at times small aphthous crusts in connection with these red patches. Abrasion of the lining membrane, like the rolling up of the mucous coat into small shreds or strings, leaving the papillæ bare for an indefinite space, is not an uncommon appearance. These diseased appearances, when very slight, do not always affect essentially the gastric apparatus; when considerable, and particularly when there are corresponding symptoms of disease, as dryness of the mouth, thirst, accelerated pulse, etc., no gastric juice can be extracted.' In other parts of his essay Beaumont mentions exudation of grumous blood and the formation of patches of aphthous ulceration as being of frequent occurrence in cases of gastric catarrh.

Acute inflammation of the stomach in children is occasionally accompanied by the formation of a false membrane.[1] This variety of the disease is most common in cases of pharyngeal diphtheria,[2] but it may also occur in other conditions. Thus, 'membranous gastritis' has been described in cases of marasmus and infantile cholera (Parrot),[3] and has been encountered after death from scarlatina (Steiner and Neureuter[4]), measles (Löschner[5]), and typhus (Vidal[6]). Wilson Fox[7] observed a similar affection of the stomach in pulmonary tuberculosis, and more recently Thomson[8] has recorded another instance of this association. In other cases, again, the disease has arisen during the course of pyæmia (Rokitansky[9]); while Rayer[10] states that it is occasionally found in cases of croup. In severe instances the whole of the inner surface of the stomach is completely covered with membrane, but as a rule the disease only occurs in the form of small patches or streaks situated in the fundus of the organ.

---

[1] 'Experiments and Observations,' 1838, p. 99.

Morbid Histology.

By means of the microscope it is possible to distinguish two varieties of inflammation of the stomach, termed respectively the *interstitial* and the *parenchymatous*, according as the disease appears primarily to affect the connective tissue or the glandular apparatus of the organ. In certain cases both of these phenomena coexist and are associated with the presence of a false membrane upon the surface of the organ (membranous gastritis).

---

[1] Woilstein, *Archiv. f. Pediat.*, 1892, vol. ix., p. 489.

[2] Author, *Path. Soc. Trans.*, vol. xlv., p. 61. Kalmus states that membrane was found in the stomach in 6·5 per cent. of his collected cases of diphtheria.

[3] *Op. cit.*

[4] *Vierteljahrschr. f. Prak. Heilk.*, 1866, Bd. i.

[5] *Jahrb. f. Kinderheilk.*, 1868, s. 330.

[6] *Bullet. Soc. Anatom.*, 1851.

[7] 'Diseases of the Stomach,' 1872, p. 115.

[8] *Archiv. f. Pediat*, 1895, s. 286.

[9] 'Path. Anat.,' *New Syd. Soc. Trans.*

[10] 'Dict. de Méd.,' vol. x., p. 124.

Acute Interstitial Gastritis.—In this variety the principal signs of inflammation are to be found in the connective tissue which binds together and supports the glands of the mucous membrane. The disease usually arises from local irritation of the stomach by the products of food fermentation, and its effects are most pronounced in the pyloric region near the lesser curvature. In the thick layer of mucus which usually adheres to the surface of the section, a large number of blood cells and masses of detached epithelium can be observed. The superficial epithelium is either shed at an early period of the disease, or its component elements are converted into cells of the goblet type.

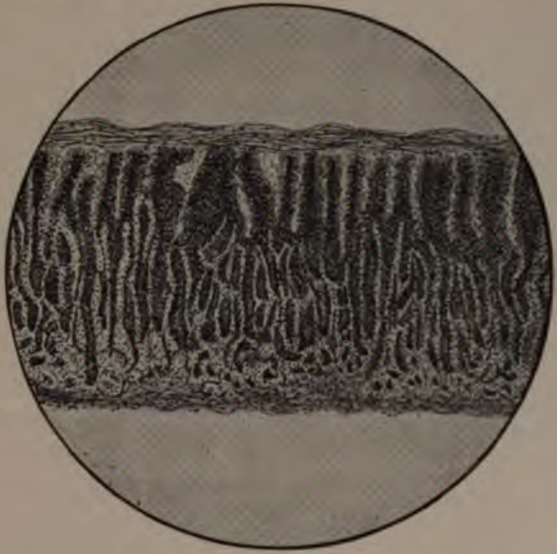

FIG. 10.—Section of the Stomach in a Case of Acute Gastric Catarrh, showing the Accumulation of Small Round Cells in the Interstitial Connective Tissue. (× 80.)

The capillaries which ramify upon the surface of the stomach and between the tubules are engorged, and here and there minute extravasations of blood may be observed. The main feature of interest consists of an accumulation of small round cells in the interstitial connective tissue. As a rule, this chiefly occurs in that portion of the mucous membrane which lies immediately below the superficial epithelium, where the inflammatory exudation tends to press upon and obscure the ducts of the glands. It may usually be noticed, however, that at its commencement the disease exhibits an irregular or patchy distribution, but that at a later date the whole of the inner coat of the stomach is affected in a similar manner. Occasionally the round-cell infiltration commences in the deeper layers of the mucous membrane around the fundi of the glands, and spreads thence toward the surface along the distribution of the interglandular bloodvessels. The exudation itself consists mainly of leucocytes, but an active proliferation of the cells of the connective tissue also contributes to the number of new morphological elements. The pressure which is thus exercised upon the parenchyma gives rise to secondary changes in the gastric glands, the fundi of which become distended with secretion, while their epithelium is flattened against the basement membrane.

The solitary glands undergo a considerable increase in size, and may even encroach upon the surface of the mucous membrane and give rise to follicular ulceration.

Acute parenchymatous gastritis is usually encountered as the result of some infectious fever or other toxæmic condition. The superficial epithelium becomes detached at an early stage, and can usually be discerned in the mucus which covers the surface of the tissue. The gastric glands present an irregular and ill-defined outline, and it is often a matter of some difficulty to distinguish one tubule from another, owing to the fact that they overlap at certain spots. The contents of the gland is mainly composed of a finely granular material, which completely fills the tubule and obliterates its lumen (Fig. 11). Here and there a nucleated cell can be faintly discerned, but the great majority of the peptic cells are unrecognisable. In many cases a granular detritus blocks the mouths of the glands and gives a characteristic black coloration when treated with osmic acid. Occasionally an elongated mass of granular material can be observed to project from one of the ducts, and similar bodies may be detected in the mucus contents of the stomach when examined with the microscope (Fig. 12).

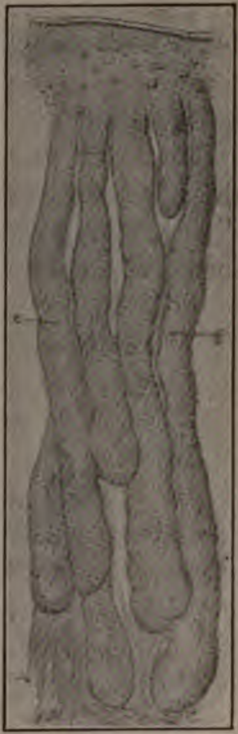

FIG. 11.—Drawing of the Glands of the Gastric Mucous Membrane in a Case of Scarlatina, showing the appearances presented by acute parenchymatous inflammation: (*f*) granular contents of tubules; (*g*) nucleated cells.

These cylindrical masses must, therefore, be regarded as casts of the gastric glands, and similar to those which occur in the urine in cases of acute Bright's disease. Little or no engorgement of the bloodvessels exists, owing to compression of the interglandular capillaries by the swollen parenchyma.

Around the fundi of the glands a certain amount of round-cell exudation can usually be observed; but this never occurs to any marked degree, and is quite a subsidiary phenomenon. When recovery takes place, the glandular epithelium is regenerated from some of the peptic cells which have escaped destruction (Fig. 13).

The false membrane which occasionally forms upon the stomach in cases of diphtheria and other specific diseases is firmly adherent to the mucous surface by means of numerous processes which pass into the ducts of the gastric glands. The superficial portion of the membrane exhibits an indefinite structure, and stains feebly with hæmatoxylin. Scattered through its substance are numerous micrococci, shreds of fibrin, and nuclei. The deeper layers of the membrane consist of numerous glistening fibres, which interlace in all directions and enclose many deeply-stained nuclei and epithelial cells. The fibres themselves hardly stain at all with hæmatoxylin or carmine, and but slightly with eosine. The morbid changes in the mucous

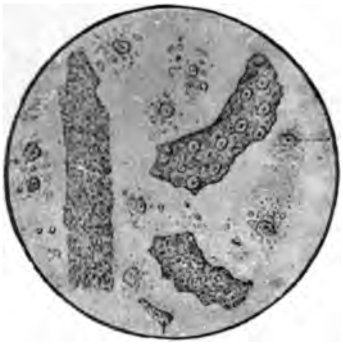

FIG. 12.—Casts of the Gastric Glands.

membrane in these cases are those of severe parenchymatous and interstitial inflammation.

CLINICAL VARIETIES AND THEIR SYMPTOMS.

Owing to the numerous ways in which gastric catarrh may arise, and the similarity which exists between the general symptoms of the disease, whatever be its exciting cause, it is impossible to offer any efficient classification of the clinical forms of catarrhal inflammation of the stomach from a consideration of their etiology. In like manner, the symptoms which accompany the different pathological varieties of the complaint are, as a rule, indistinguishable from one another, so that any attempt to delineate the clinical aspects of the disease from a study of its morbid histology is equally futile. It is necessary, therefore, to distinguish primarily between those forms of gastric catarrh which are accompanied by fever and those which are not, and to describe each subvariety of these two principal disorders in a separate manner.

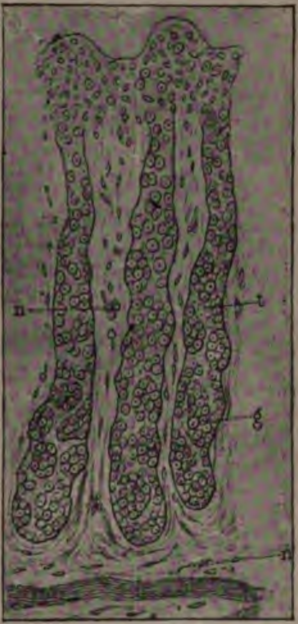

FIG. 13. — Drawing of the Mucous Membrane of the Stomach in a Case of Measles, showing the regeneration of the secretory epithelium (g) and a slight amount of interstitial exudation (n).

The Apyrexial or Non-febrile Variety.

Gastric catarrh unaccompanied by fever is one of the commonest disorders of childhood, and is usually excited by some error of diet. Hereditary predisposition is a well-marked feature in these cases, for in some families the least indulgence in certain articles of diet is at once followed by symptoms of the complaint. There are four varieties of non-febrile gastric catarrh which appear to be worthy of special notice, namely:

1. Acute indigestion, or 'embarras gastrique.'
2. Subacute gastric catarrh, or 'biliousness.'
3. Recurrent catarrh, or 'cyclical vomiting.'
4. Toxic gastritis.

1. *Acute indigestion*, or, as it is termed by French writers, 'embarras gastrique,' usually commences without any premonition—indeed, in many instances the general health is especially good and the spirits remarkably buoyant immediately prior to an attack. In other cases the onset of the disorder is preceded for several days by anorexia, headache, and other symptoms of malaise. The complaint may begin at any time in the day, but as a rule it commences in the early hours of the morning. In such cases the child falls into a heavy sleep soon after going to bed, but a few hours later becomes restless and talkative. As the morbid condition of the stomach develops, it appears to affect the cerebral convolutions in a reflex manner, without the source of the peripheral irritation being localized, so that towards morning the sleep becomes disturbed, and is accompanied by a series of horrible dreams, in which the child usually pictures itself being

pursued by wild animals or fighting for its life. When the epigastric discomfort has attained a certain degree of intensity the child awakes with a feeling of nausea or giddiness, and vomits as soon as it attempts to get out of bed. The ejecta are considerable in quantity, have a sour taste and smell, and consist of the food taken at the last meal, the various ingredients of which have undergone little or no digestive changes. A considerable quantity of lactic acid is usually present in the fluid obtained by filtration of the vomit, but free hydrochloric acid is invariably absent, and the fluid itself is devoid of digestive properties even after acidification. Although the evacuation of the stomach usually affords relief, the nausea soon returns, and is accompanied by an excessive secretion of saliva and a sensation of faintness. The pulse is weak, fluttering, and often abnormally slow; the extremities are cold and covered with sweat; and the temperature of the body is subnormal. The face is pale, and the eyes and mouth are surrounded by dark rings. In many cases severe frontal headache supervenes, and is accompanied by intolerance of light and sound. These symptoms culminate in violent retching and vomiting, which recur at short intervals, and result in the expulsion of a quantity of glairy, alkaline mucus. In many cases bile regurgitates into the stomach, and imparts a characteristic yellow tinge and bitter taste to the ejecta. Occasionally fits of shivering, or violent pain in the perinæum, follow each attack of emesis. The child expresses a loathing for food, and any attempt to force it to partake of nourishment immediately gives rise to vomiting. The tongue is coated with a moist, creamy fur, the breath is offensive, and thirst is usually a marked symptom. In some cases the irritant contents of the stomach find their way into the intestine, and set up colicky pain in the abdomen, and acute diarrhœa. In young children epileptiform convulsions may ensue from exhaustion of the nervous system. The attack may only last for an hour or two, or it may persist for twenty-four to forty-eight hours, those which commence in the early morning being the most severe and of the longest duration. When improvement sets in, the attacks of emesis gradually become less frequent, and finally the patient falls into a heavy sleep, from which he awakes free from nausea and headache. This, however, does not necessarily indicate that the catarrhal process in the stomach has quite subsided, for in most cases where an attack has proved severe, the child suffers for several days from malaise, want of appetite and discomfort after food, and is liable to a return of the symptoms from trivial causes. In certain cases the voice continues hoarse for several days after each attack.

Although acute indigestion arising from catarrh of the stomach is usually accompanied by the symptoms already described, in not a few cases the ingestion of substances of an irritant or indigestible nature is followed by nervous phenomena of a severe and puzzling character. Thus, in some cases the initial symptoms of the complaint consist of extreme apathy and drowsiness, or the child may suffer from giddiness, noises in the ears, and dangerous cardiac failure for several hours before the nausea commences. In other cases, again, dimness of vision, strabismus, or hemianopia have been observed to precede the gastric attack. Acute delirium with hallucinations also occurs in a small proportion of the cases. Occasionally aphasia constitutes the first symptom of the disease. Thus, Henoch[1] relates the case of a boy, aged three years, who was suddenly attacked with speechlessness, and could only utter a sound like 'au' when the skin was pinched. The eyes were prominent and staring, but otherwise nothing abnormal could be discovered. About an hour and a half after the appearance of the aphasia he suddenly vomited several cherries, when the power of speech was immediately restored. An almost similar case in a girl nine years old has been recorded by Siegmund.[2] Sometimes impairment of speech is associated with a state of mental aberration or depression, as in another case related by Henoch, where a boy, six years old, became suddenly aphasic and semi-unconscious after a heavy meal of fruit and cakes. These symptoms disappeared as soon as the bowels had been evacuated.

In other instances, acute irritation of the stomach finds its expression in a series of reflex spasms or convulsions. Thus, it is by no means uncommon to observe seizures of an epileptiform nature follow the ingestion of indigestible substances in children of a nervous and excitable temperament. In this connection Henoch[3] quotes the case of a boy who was attacked by vomiting after indulgence in a heavy meal, and on the next day had three epileptic fits, which were followed by coma. This latter condition persisted for three days, at the end of which time the boy regained consciousness, but continued aphasic. Complete recovery ensued within ten days.

---

[1] 'Diseases of Children,' *New Syd. Soc. Trans.*, vol. i., p. 171.

[2] *Berl. Klin. Woch.*, 1883, s. 335.

[3] *Op. cit.*, p. 170.

Paralysis is rarely encountered as the result of 'embarras gastrique,' so that a case recorded by Fränkel possesses an unusual degree of interest. The subject was a girl, four years of age, who was attacked, half an hour after a heavy meal, with right hemiplegia and anæsthesia, the right cornea being insensitive. Sensation was restored the following day, and the paralysis rapidly passed off. Functional affections of the heart are also occasionally observed. Thus, in some cases the pulse does not exceed forty or fifty beats per minute, and is apt to be irregular and intermittent. The depression of the heart's action may continue for several hours, and is often accompanied by extreme pallor of the face, vertigo, and oppression at the precordium. These several symptoms rapidly subside after vomiting has taken place.

Another series of reflex phenomena arising from gastric irritation are connected with the respiratory organs. A sense of tightness at the chest associated with an inspiratory form of dyspnœa, or excessive gasping or yawning, is a frequent symptom of acute indigestion, while Henoch, Silbermann, and others, have described a variety of asthma (asthma dyspepticum) attributable to the same cause. In cases of this kind the child is attacked with urgent dyspnœa, accompanied by cyanosis, coldness of the extremities, and a quick, small and irregular pulse, all of which symptoms disappear immediately the stomach has rid itself of its irritating contents.

2. *Subacute catarrh* of the stomach, popularly known as 'biliousness,' is a very common complaint between the ages of four and sixteen. In many cases

the disorder is excited by the abnormally rich and stimulating diet with which children are often supplied by indulgent and foolish relatives; but in some instances no such cause can be assigned for the origin of the complaint. The subjects of this disease suffer from chronic depression of the spirits, and are disinclined to any form of physical or mental exertion. They usually present a pale or sallow complexion, with dark lines beneath the eyes, and a slight icteric tinge in the conjunctivæ. During the greater part of the day they mope about the house, declining to go out on the score of fatigue, or to read on account of headache. In some cases the speech is hesitating, or the child stammers and becomes inarticulate when asked a question. In other instances the patient acquires a habit of frowning, winking the eyes, or making sudden grimaces, while the limbs are constantly jerked about, so that he cannot sit on a chair for a minute without shifting the position of his feet. The temper is extremely uncertain, and often peevish, while at night the sleep is disturbed by horrible dreams. Talking in the sleep, grating of the teeth, and incontinence of urine, are also frequently observed. In most instances constant and severe headache is a marked feature of the complaint. The pain is usually localized to the frontal region, but often radiates along the course of one or other supra-orbital nerve. In some cases the skin over the affected region is distinctly hyperæsthetic. The headache is most severe in the early morning or after the mid-day meal, and is increased by active movements of the head, by stooping the body, and by the use of alcohol. Epigastric discomfort after meals is seldom absent, and is most marked in the evening, when the abdomen becomes distended from intestinal flatulence. As a rule, the appetite is much diminished, and the child expresses the greatest loathing for fat and sweet articles of diet; but occasionally the desire for food is excessive. In the early morning the tongue is coated with a thin white fur, through which the papillæ can be faintly discerned, but towards mid-day it often becomes clean and abnormally moist. In some cases the dorsal aspect of the organ presents a curious appearance of superficial ulceration, which takes the form of oval circumscribed patches of a reddish colour, situated in a symmetrical manner upon either side of the median line, or occupying the centre of the posterior third. This morbid condition is dependent upon an exfoliation of the superficial layers of the epithelium, and is seldom attended by pain. In some cases small irritable ulcers make their appearance upon the sides of the tongue or upon the mucous membrane of the buccal cavity, and occasion considerable discomfort during the process of mastication. They are apt to recur with each attack of the gastric disorder, and are very difficult to cure. Nausea is a common symptom, and often persists for many days, but vomiting only occurs at intervals. Heartburn and eructations of fœtid gas are a source of frequent complaint after meals, and the child often yawns incessantly, gasps for breath, or suffers from hiccoughs or 'goose-skin' during the periods of digestion. Extreme thirst is sometimes observed, and a strong inclination may be evinced for acid drinks. The bowels are usually confined, and the stools are hard, clay-coloured, and passed with pain and straining. Occasionally the complaint is preceded by the passage of a large quantity of limpid urine, but as soon as the disorder becomes established, the urinary secretion is diminished in amount, and deposits lithates or uric acid upon standing. The pulse is small and feeble, and the patient often complains of palpitation, or a sense of faintness after meals, and may even suffer from actual syncope. In some cases each attack is preceded and accompanied by urticaria or herpes.

The duration of the disease is uncertain. When appropriately treated, it usually terminates in a few days, though a weak condition of the digestive organs may persist for some time afterwards (see Chapter VIII.). In neglected cases, or when food is indulged in as usual, the disorder may be prolonged for many weeks, and give rise to severe prostration and inanition. Occasionally the symptoms of acute gastric catarrh supervene during the course of the subacute disease.

3. *Recurrent Catarrh.*—Under the various titles of 'periodic,' 'fitful,' 'recurrent,' and 'cyclical' vomiting, different writers have described a disease of childhood characterized by severe attacks of emesis, which exhibit a tendency to periodic recurrence. Cases of this description are extremely common in practice, and are in reality merely examples of a variety of gastric catarrh, which in certain individuals has a tendency to recur from slight and ill-defined causes. In some instances a tendency to the complaint appears to have been transmitted from parent to child for several generations, but in others no such history can be obtained, although several members of the family may have suffered from the disease in early life. The disorder usually commences between the ages

of two and five, and, according to my experience, is more common in boys than girls. In some cases the first attack appears to follow exposure to cold or indulgence in some special article of food, while in others the disease originates spontaneously during the convalescent period of some infectious fever, such as measles or scarlatina; but whatever the original cause may have been, the complaint, when once established, exhibits a peculiar tendency to recur at short intervals, and without apparent reason. In some children, however, any slight disturbance, such as fatigue, anger, a change of diet, or even a shift of the wind toward the east, is immediately followed by the symptoms of the disorder.

Although the attacks exhibit a certain periodicity of recurrence, it must not be supposed that they develop at regular intervals of time. On the contrary, the disorder is usually extremely uncertain in its onset, at one time recurring once or twice a week, while at another several weeks, or even months, may elapse between the consecutive attacks. As a rule, the most obstinate and severe cases are those which date their origin from the convalescent period of measles or typhoid. In such instances, a month rarely passes without an attack being witnessed, while in many cases two, or even three, may occur within the same space of time. With the approach of puberty, the incidence of the disease becomes less and less frequent; but even after this age the subjects of this disorder are prone to develop catarrh of the stomach from trivial causes.

In its clinical aspect this variety of gastric catarrh does not differ materially from the acute form already described. It commences at any time of the day, more especially in the early morning or after the midday meal. As a rule, nausea and vomiting are the first symptoms to attract attention; but occasionally the child suffers from headache or abdominal pain for several hours before the emesis commences. The face is pale, the pulse small and feeble, and occasionally dimness of vision and giddiness are complained of. The first effect of the vomiting is to rid the stomach of the remains of the last meal, which from its large quantity and undigested appearance affords conclusive proof that the motorial and secretory functions of the organ had been in abeyance for some time. Further examination shows that the material is either neutral or faintly acid in reaction, and that hydrochloric acid is absent. Occasionally I have detected traces of lactic acid in the vomit, but this is seldom present unless the food has undergone stagnation for several hours. Although the first act of emesis may afford temporary relief, the sensation of nausea soon recurs, and is followed by severe retching. From time to time small quantities of mucus mixed with saliva are rejected, and occasionally streaks of blood may also be observed. In severe cases the vomit may consist entirely of bilious fluid. During the course of the disorder the bowels are confined, and the tongue presents a uniform coating of thin white fur. Between the attacks of retching the child exhibits mental and physical depression of a severe kind, and lies motionless upon the bed, apparently unable even to turn over or lift up its head. Nevertheless, there exists but little danger to life, for recovery takes place rapidly as soon as the gastric intolerance subsides. In the vast majority of cases the temperature of the body remains subnormal as long as the vomiting persists; but occasionally, and especially in children debilitated by previous disease, a slight degree of fever may be observed towards evening.

It is seldom that any special nervous phenomena accompany an attack, but in more than one case I have known strabismus, associated with intolerance of light, to lead to the diagnosis of meningitis.

The duration of the disorder varies in different cases, the extreme limits being two hours and three days. Recovery is usually rapid, but between the attacks the child remains pale, thin, and irritable, and often suffers from want of appetite, with constipation and clay-coloured stools

4. *Toxic Gastritis.*—When the inflammatory condition results from the ingestion of irritant poisons, the symptoms of the disease differ materially from those which attend the simpler varieties of the complaint. Children seldom swallow mineral poisons intentionally, but they occasionally partake of putrid meat, or food which has become accidentally contaminated with arsenic or copper. In most of these cases it is possible to distinguish between the immediate and remote effects of the poison, or, in other words, between the symptoms due to direct irritation of the stomach and those which ensue at a later period from the action of the poison upon the central nervous system and other tissues. Among the *immediate* symptoms of toxic gastritis, the most important are pain at the throat and epigastrium, extreme thirst, which is not relieved by drinking water, incessant retching, and vomiting of mucus or blood. The pulse is small, feeble, and

often irregular; the face pale and anxious, the lips cyanosed, the temperature subnormal, and the trunk and extremities bathed in a profuse perspiration. The urine is diminished in quantity, or even suppressed; the respiratory movements are superficial and rapid, and occasionally several liquid motions containing altered blood are evacuated in rapid succession. The abdomen is distended, and very tender in the epigastric and umbilical regions. In this state the patient may succumb within a few hours from collapse, or death may occur after a day or two from asthenia or peritonitis.

The *secondary* or *toxic phenomena* of the disease consist of albuminuria, jaundice, hæmorrhages into the skin or from the various mucous membranes, paresis of the voluntary muscles, an enfeebled action of the heart, or severe inflammation of the stomach and intestine accompanied by vomiting and diarrhœa. In cases of poisoning by putrid meat, the onset of symptoms may be deferred for several hours. Colicky pain in the abdomen, accompanied by a loose action of the bowels, is usually the first indication of the complaint; but nausea and vomiting soon make their appearance, and persist throughout the whole course of the disease. The ejecta seldom contain blood, but the stools are often black and exceedingly fœtid. The subsequent toxic phenomena consist of gradual failure of the heart with cyanosis. Death usually results from syncope or asthenia, and sometimes occurs quite suddenly long after all danger appears to have passed away.

Even when the patient escapes both the immediate and remote effects of the poison, recovery is usually tardy and imperfect. In many cases the symptoms of gastric inflammation persist for a long time, and the mucous membrane of the stomach undergoes changes of an organic nature. As the result of this, the general nutrition suffers, and the patient eventually succumbs to pulmonary tuberculosis[1] or to some other intercurrent disease.

The Febrile Form.

In this variety of gastric catarrh the symptoms commence suddenly, with a rigor and pains in the back and limbs. Occasionally the disease is ushered in with a convulsion, or with severe headache and vomiting. In all cases the temperature rises rapidly, and may reach 103° or 104° F. by evening, when the child is apt to become slightly delirious. Vomiting is an inconstant symptom, but in some cases repeated emesis occurs, with the rejection of bile and mucus. The bowels are usually confined; but if the catarrhal affection attacks the mucous membrane of the intestine as well as that of the stomach, the child may suffer from colicky pains in the abdomen, accompanied by watery and offensive diarrhœa. When the duodenum is inflamed, jaundice often results. The tongue is at first thickly coated upon the dorsum, while its edges and tip are clean and red. In severe cases the organ becomes red and fissured after a day or two, the lips dry and cracked, and sordes may appear on the teeth. The appetite is completely lost, but thirst is usually severe. Frontal headache, restlessness, and depression continue throughout the attack. Sleep is disturbed, and delirium often occurs towards evening. The pulse is quick, weak, and compressible. The fever is distinctly remittent in character, the temperature rising to 102° to 104° F. at night, and falling to 99° or 100° in the morning. It usually attains its maximum about the second or third day, after which it gradually declines, becoming intermittent and finally subnormal. Occasionally the disease terminates by crisis, the temperature falling several degrees in the course of a few hours, while its defervescence is accompanied by profuse sweating, diuresis, or by a sharp attack of diarrhœa. During the whole febrile period the urine is diminished in quantity, and presents a copious deposit of lithates on standing. In some cases the addition of a few drops of the tincture of perchloride of iron gives rise to a blood-red colour in the urine, and to a copious white deposit. In these cases the breath has a sweet odour, like that observed in diabetes. These phenomena depend upon the formation in the stomach of some substance allied to acetone, notwithstanding the contrary opinion expressed by Savelieff[1] and Baginsky.[2] As a rule, the complaint lasts between seven and ten days, but if the stomach is irritated by the administration of stimulants or tonics, it may persist for a much longer time.

---

[1] Author, *Clin. Soc. Trans.*, vol. xxvii., p. 69.

When gastric catarrh occurs in an epidemic form during the prevalence of typhoid or cholera, the complaint exhibits a somewhat different aspect. The temperature remains elevated for ten days or a fortnight, and abdominal pain and diarrhœa are usually present. This variety of gastro-intestinal catarrh subsides in a gradual manner, and is not infrequently followed by one or more relapses. During the progress of the disorder the child loses flesh and colour, and may subsequently suffer from feeble digestion for many months. Cases of this description are often termed 'gastric fever' or 'abortive typhoid,' and apparently depend upon the introduction of some modified form of the typhoid bacillus.

---

[1] *Berl. Klin. Woch.*, 1894, s. 754.

[2] *Archiv. f. Kinderheilk.*, ix., s. 1.

Membranous gastritis is seldom accompanied by any special symptoms. Occasionally aversion to food, nausea, vomiting, or tenderness of the epigastrium in cases of diphtheria, suggest the possibility of a secondary affection of the stomach; but the only positive sign of the complaint consists in the rejection of pieces of membrane. In a case of tuberculosis recorded by Thomson[1] a complete cast of the stomach was vomited.

PHYSICAL EXAMINATION.

Examination of the abdomen seldom affords any important indication as to the existence of gastric catarrh. In severe cases, the epigastric region may be somewhat distended and tender, while in those cases where catarrh of the bowel co-exists there may be general distension, with pain on pressure over the umbilical and right iliac regions. The spleen is never enlarged, nor can any dilatation of the stomach be detected.

Chemical examination of the vomit reveals an absence of both hydrochloric acid and pepsin. Mucus is always present in excess, and when examined with the microscope it is found to contain a large number of epithelial cells, salivary corpuscles, cocci, and bacteria. In the parenchymatous form of inflammation, such as occurs in scarlatina and renal disease, casts of the gastric glands may often be discovered. In certain cases, small pieces of the mucous membrane of the stomach become detached as a result of the inflammatory disease, and may be recognised in the vomit or in the washings of the organ. The occurrence of membrane in gastritis has already been referred to.

DIAGNOSIS.

The non-febrile variety of the disease does not usually present any difficulty of diagnosis. In severe cases, however, where retching or vomiting persists for several days, and the child suffers from headache, accompanied by slight strabismus, the question of meningitis may possibly arise. But in such cases inquiry will usually elicit the fact that the child has previously suffered from attacks of a similar nature, and this, combined with the absence of fever and of the other symptoms of inflammation of the meninges, is sufficient to indicate the real nature of the complaint.

The febrile form is less easy of immediate diagnosis, owing to the fact that most of the infectious diseases of childhood are accompanied at their onset by symptoms of gastric catarrh. In these cases, however, a correct opinion can be formed as soon as the rash or some other characteristic symptom develops. The main difficulty is to distinguish between gastric catarrh and mild cases of enteric fever, and in some instances several days must elapse before any definite opinion can be given. It may be noticed, however, that in enteric fever the temperature usually rises gradually, and does not attain its maximum for several days, while the diurnal remissions are unimportant Diarrhœa, with loose yellow stools, is the rule, and vomiting the exception. The abdomen becomes uniformly distended, and pain or gurgling may be elicited upon pressure over the cæcum. The spleen enlarges, and about the end of the first week the eruption appears upon the abdomen and back. In gastric catarrh, on the other hand, the fever attains its maximum on the second or third evening, and the temperature falls several degrees each morning. Diarrhœa is an infrequent symptom, but nausea, heartburn, or flatulence is apt to occur after each meal Herpes appears upon the lips in many cases, and in not a few catarrh of the nasal and respiratory passages complicates the gastric disorder. The spleen never enlarges, and there is no characteristic eruption.

TREATMENT.

General. — In all inflammatory diseases of the stomach, rest in the recumbent position is an imperative necessity. When the complaint is accompanied by fever, the child should be confined to bed for the first few days, and only permitted to get up when the temperature has become intermittent. In less severe cases, the patient may dress and lie upon a couch until the gastric symptoms have subsided. If abdominal pain proves severe, the epigastric region should be covered with a large linseed poultice, or frequently fomented with hot flannels. Leeches and dry cupping are seldom required. When convalescence has set in, a change of air is often of great value. In this connection it is worthy of notice that sea-air often induces a recurrence of the gastric disease, so that some inland health resort, like Malvern or Ilkley, is to be preferred to one situated upon the coast. In all cases, the clothing should be warm, and a flannel bandage may be worn round the waist during the winter months. Fresh air and exercise are indispensable in the preventive treatment of the disease.

Dietetic.—As long as the vomiting continues urgent, no attempt should be made to administer food, but the patient may be allowed to wash out the mouth with warm water, or to suck small pieces of ice from time to time. As a rule, the subjects of acute gastric catarrh can forego every kind of nourishment for twenty-four hours without ill effects; but very young or debilitated children are apt to exhibit signs of exhaustion after a few hours, and in such cases nutrient enemata, containing a small quantity of stimulant, may be given occasionally. As soon as the more urgent symptoms have subsided, a cautious trial can be made of small doses of milk and soda-water, and if this is retained by the stomach, cold Bovril, or chicken or veal broth, may be administered. In some cases, the persistence of the nausea after the gastric intolerance has abated is due to exhaustion, and can be best combated by the use of a little good brandy or champagne. Occasionally milk gives rise to flatulence or vomiting, and requires to be peptonized before its administration. Solid food may usually be given within forty-eight hours of the subsidence of the gastric symptoms, but the diet must be carefully regulated, and excess of food at meal-times prohibited.

In the febrile disorder, a liquid diet should be strictly enforced until the temperature commences to intermit, after which milk-puddings, bread-and-milk, and toast may be allowed. When the febrile symptoms have quite subsided, the child may gradually resume its former mode of living; but it must be remembered that a weak state of digestion usually persists for many weeks after an attack of gastric catarrh.

To prevent a recurrence of the complaint, the diet should be strictly regulated, to the exclusion of those articles of food which are prone to undergo fermentation in the stomach. Indulgence in sweets and cakes between meals must be prohibited, and the child should be taught to spend a certain time over each meal, and to chew each mouthful of food well before swallowing it. If proper mastication is impossible on account of a bad state of the teeth, the latter must receive immediate attention.

Medicinal.—At the commencement of an attack, an attempt should be made to empty the stomach of any undigested food which it may contain. With this object, a full dose of ipecacuanha, sulphate of zinc, or sulphate of copper, may be administered at once (p. 85). If emesis has already occurred, the child should be encouraged to drink one or two tumblersful of warm water, and afterwards to provoke vomiting by thrusting a finger down the throat. In this manner the stomach can be thoroughly cleansed, without recourse being had to the more objectionable process of lavage. If the case is seen for the first time after the organ has been completely evacuated, the chief indication is to rid the intestines of the irritant material they contain. For this purpose the various preparations of mercury are invaluable. As a rule, the best results are obtained by the administration of one or two grains of calomel every four hours, but occasionally a dose of blue pill, of gray powder, or of the compound calomel pill, is equally advantageous. Many practitioners prefer to give castor-oil or some other purgative draught at this stage of the complaint; but this procedure is apt to increase the vomiting. As soon as the retching shows signs of abatement, frequent small doses of the effervescing citrate or sulphate of magnesia may be given, or the compound senna mixture or decoction of aloes may be administered every four hours until free purgation has been induced. In those cases where retching continues for more than twelve hours, it may be necessary to employ a sedative in order to relieve the irritability of the stomach. For this purpose an effervescing mixture, containing a few drops of the solution of morphine, combined with carbonate of sodium, carbonate of bismuth, and hydrocyanic acid, may be tried. In the most severe cases, it may be necessary to give a hypodermic injection of morphine.

In the febrile form of the disease, where gastric symptoms are seldom severe, frequent small doses of calomel, with the occasional use of an effervescing alkaline medicine, are usually all that is required.

In the subacute variety of the complaint, mercurials are of the greatest value, more especially calomel and gray powder. One or other of these may be administered each night for a week, while during the day a mixture containing bicarbonate of sodium, taraxacum, and rhubarb, may be given between the meals.

When the catarrh of the stomach has subsided, it is a mistake to prescribe strong tonics with the view of improving the general health. Quinine and iron almost always disagree, and their administration frequently occasions a relapse of the complaint. It is much wiser to continue with an alkaline medicine, to which nux vomica, calumba, chiretta, or gentian can be added if necessary. Pepsin, along with dilute hydrochloric acid after meals, is of great value in cases where flatulence and loss of appetite persist for a long time after the subsidence of the immediate symptoms of the disease.

In that form of gastric catarrh which has a tendency to return every few weeks (recurrent catarrh), I have found the administration of a dose of the sulphate and carbonate of magnesia in half a tumblerful of warm water each morning before breakfast to be of especial value in preventing a relapse—indeed, out of the very large number of cases which have come under my care, I have hardly ever known this simple remedy to fail.

SCHEME OF DIET IN SUBACUTE GASTRIC CATARRH.

The best diet for your child is:

For Breakfast at 8 *a.m.*—Milk, peptonized or diluted with one-third of its volume of lime-water; weak tea with milk, cocoa made from the nibs; stale bread, toast, plain biscuits or rusks, with a scrape of butter; bread-and-milk.

Avoid: Coffee, sweet cocoa, strong tea, cream, sugar, much butter, new bread, cakes, pastry, eggs, jams, marmalade, preserved fruits, porridge.

For Dinner at 1 *p.m.*—Soups made without vegetables, beef-tea, beef essence, Bovril, mutton, chicken or veal broth, calf's foot jelly, a small quantity of finely-minced chicken, plaice, or sole; stewed celery, asparagus, sea-kale; sago, tapioca or vermicelli puddings made with milk; a glass of hot water, soda-water, toast-water, freshly-made lemonade.

Avoid: Strong soups, mackerel, salmon, cod, beef, rabbit, pork, veal, potatoes, cabbage, parsnips, pastry, meat pies, spices, pickles, condiments, sweets, custards, fruit of all kinds, beer, spirits, wines.

For Tea at 5 *p.m.*—Same as breakfast.

For Supper at 7.30 *p.m.* (*if necessary*).—Milk, cocoa made from the nibs, bread-and-milk, milk-puddings.

General Rules.

1. A flannel bandage should be worn round the waist next the skin. Exposure to cold winds must be carefully avoided.

2. Open-air exercise should be taken every day. Gymnastics, horse-riding and cycling are of great value.

3. The surface of the body should be sponged each morning with tepid salt-and-water, and friction afterwards applied by means of a coarse towel.

CHAPTER VII.

CHRONIC GASTRIC CATARRH IN CHILDREN.

Chronic catarrh of the stomach is much less common in childhood than during the period of infancy. When it occurs as a primary disease, it usually attacks ill-nourished and debilitated children, or those who are already suffering from rickets, syphilis, or scrofula. Occasionally the first symptoms follow an attack of measles or whooping-cough. The exciting cause of the complaint is usually to be found in the habitual administration of rich or indigestible foods; but sometimes the use of gin or other alcoholic liquors is directly responsible for the gastric disease. Thus, in two cases of cirrhosis of the liver in children which recently came under my care, microscopical examination of the stomach after death revealed well-marked chronic catarrh of the mucous membrane. A septic state of the mouth, arising from sloughing of the gums or a carious state of the teeth, is occasionally responsible for the symptoms of the disease; while the excessive use of tobacco at an early age must also be recognised as a cause of the complaint.

A large proportion of the cases of chronic catarrh of the stomach which are met with in practice are the result of disease of the kidney, liver, or heart; while in not a few instances the disease is secondary to tuberculous mischief in the lungs. These cases will be considered in greater detail when the subject of dyspepsia arising from disease of organs other than the stomach is discussed (Chapter XII.).

In many cases a catarrhal affection of the bowel accompanies the disease of the stomach, and gives rise to diarrhœa and other symptoms indicative of intestinal indigestion.

MORBID ANATOMY.

The naked-eye appearances of chronic gastric catarrh vary considerably in different cases. As a rule the organ is dilated, and in long-standing cases its lower border may extend for some distance below the level of the umbilicus. The most conspicuous examples of this are to be found in cases of chronic phthisis, or of lardaceous disease of the abdominal organs arising from caries of the hip or spine. The inner surface of the stomach is usually covered with a thick layer of mucus, which is particularly abundant and tenacious in the region of the pylorus. After cleansing its surface, the mucous membrane is found to present a dead-white, swollen and opaque appearance, with here and there a brown stain, due to an antecedent hæmorrhage. Should the patient have suffered from a subacute attack of inflammation shortly before his death, small areas of capilliform congestion may be observed in the fundus of the organ and along the lesser curvature. On the other hand, if the venous circulation through the stomach was greatly embarrassed during the latter end of life, the mucous membrane may exhibit a deep purple colour, with numerous punctiform hæmorrhages scattered about the cardiac region and along the greater curvature.

In those rare cases where atrophy of the secretory structures has taken place, the walls of the stomach are unusually thin and transparent, and the inner coat often presents a number of glazed or shining streaks, which run parallel with the small curvature. In this latter condition, mammillation of the mucous membrane is sometimes to be observed in the neighbourhood of the pylorus.

MORBID HISTOLOGY.

By means of the microscope it is possible to recognise the two principal forms of gastritis already described, namely, the parenchymatous and the interstitial; but the distribution of the disease is far less exact than in the acute form of the complaint.

1. *Chronic Parenchymatous Gastritis.* — In the first stages of the complaint the appearances presented by the gastric glands are identical with those already described in cases of acute inflammation of the stomach (Fig. 11). The tubules are swollen and irregular, and are so closely set together that their borders frequently overlap and quite obscure the interglandular bloodvessels. The distinction between the parietal and central cells is lost, and the proliferation of the latter, aided by an exudation of leucocytes and red corpuscles, causes the lumen of the gland to be obliterated. The second stage in the process is characterized by degenerative changes in the contents of the tubules. At first the cells lose their individual

outlines and become welded together in the form of irregular masses, which present a granular aspect and stain deeply with logwood. At a later period minute refractile globules make their appearance in the deeper portions of the gland, and among the masses of degenerated cells. These particles rapidly increase in size and number, so that eventually the tubules are filled with globules of fat which stain black with osmic acid (Fig. 14).

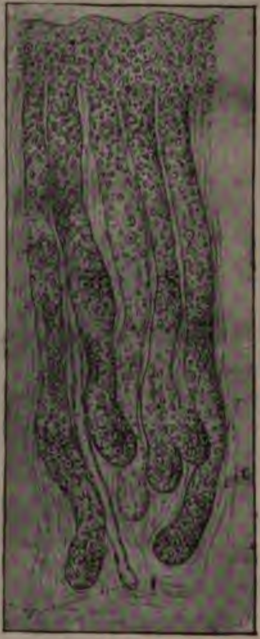

FIG. 14.—Drawing of the Gastric Glands in a Case of Chronic Parenchymatous Gastritis, showing fatty degeneration of the secretory epithelium (*t*), with thickening of the interstitial connective tissue (*v*); section stained with osmic acid.

In the last stage of the process the degenerated contents of the tubules gradually disappear, so that the gastric glands eventually resemble wrinkled membranous sacs, in which a few epithelial cells may occasionally be discerned. The interstitial tissue is much increased in amount, and the interglandular bloodvessels often exhibit distinct thickening of their inner and middle coats (Fig. 15).

2. *Chronic Interstitial Gastritis.*—The round-cell exudation which occurs during the early stages of the disease presses upon and obscures the gastric tubules. At a later period, when fibrous tissue forms between and around the glands, it tends to exert traction upon the ducts and to obliterate the orifices of the tubules. As the result of this obstruction, the deeper

portions of the glands become distended by their own secretion, and present the appearance of small cysts in the substance of the mucous membrane. The secretory epithelium then commences to undergo a series of secondary changes. The protoplasm loses its granular aspect, and becomes clear and unaffected by the ordinary dyes; while the cells themselves change from a polygonal to a columnar shape. In this manner a retention cyst is formed, which eventually becomes lined by a single layer of columnar or

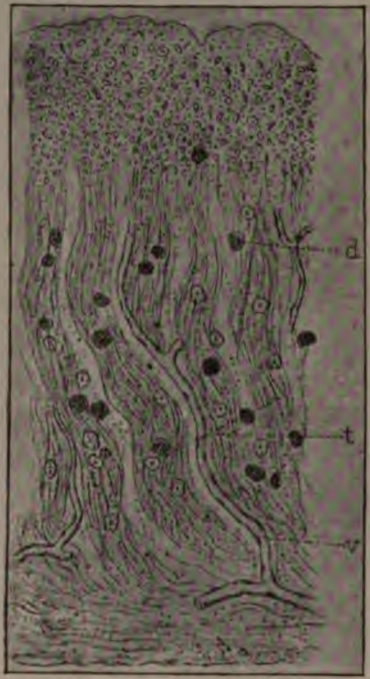

FIG. 15.—Drawing of the Mucous Membrane of the Stomach in an Advanced Case of Parenchymatous Gastritis. The glands appear as thin membranous tubes (*t*), containing a few epithelial cells (*d*), and the walls of capillary blood-vessels are much thickened (*v*).

cubical epithelium. In these cases the inflammatory process seldom extends to the submucous coat, but the muscularis mucosæ often suffers severely. Occasionally the surface of the stomach exhibits a peculiar papillary or villous appearance, which at first glance causes the tissue to resemble a section of the small intestine. These spurious villi are due to a prolongation of the interstitial tissue between the mouths of the ducts, and consist entirely of nuclei, with a few spindle cells and newly-formed capillary vessels. Finally, the excessive growth of fibrous tissue between the glands produces atrophy of the parenchyma; but even in advanced cases the disease seldom exhibits a uniform distribution.

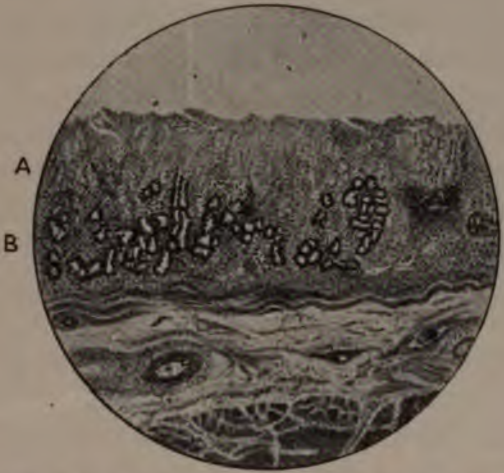

FIG. 16.—Section of the Stomach in a Case of Chronic Interstitial Gastritis, showing the formation of fibrous tissue in the mucous membrane (A), with cystic disease of the glands (B). (× 50.)

SYMPTOMS.

At the commencement of the disease the symptoms arising from maldigestion of the food only make their appearance from time to time, or after an unusually copious meal. Soon, however, *epigastric discomfort* is regularly experienced after lunch or supper. As a rule, the morbid sensations complained of are those of weight and fulness, rather than of actual pain. About half an hour after the meal the lower part of the chest feels as though it was constricted by a tight band, or a heavy load seems to rest upon the sternum or between the shoulders, and to obstruct the breathing. These symptoms increase in severity until the patient is often forced to loosen the clothes in order to obtain relief. Flatulent *eructations* occur at intervals, and not infrequently large quantities of foul gas are passed per rectum. *Pyrosis* is seldom absent, and the regurgitation of mouthfuls of acid fluid gives rise to a cramping pain behind the lower end of the sternum, or in the throat. In many cases *nausea* occurs constantly after each meal, and may also be experienced in the early morning before breakfast. *Vomiting* is an inconstant symptom, but it is occasionally observed after a heavy meal, or in the morning when the child rises from bed. In the former case the ejecta consist of undigested and fermenting food, in the latter of stringy, alkaline mucus mixed with saliva. When the dyspeptic symptoms prove severe, *reflex phenomena*, due to disturbance of the cerebral circulation, such as pain and throbbing in the head, flushing of the cheeks and ears, giddiness, or difficulty of articulation, are often observed. In some cases, palpitation of the heart or an inspiratory form of dyspnœa occurs regularly after the mid-day meal, or the child suffers from stupor and mental depression during the afternoon.

These symptoms usually last from one to three hours, and gradually subside before the next meal. If they occur during the night, the patient may suffer severely from insomnia. In the early stage of the disease the *appetite* is preserved, and is often ravenous, so that the child never appears to be content, even after a full repast. In other cases it proves extremely capricious, and the patient evinces great distaste for certain articles of diet, notably for fats and sweets. With the progress of the complaint the desire for food completely disappears, and may be replaced by a profound loathing for every kind of nourishment.

Thirst is usually exaggerated, and is most severe between the meals. Occasionally the desire for water is excessive, and almost impossible to satisfy. The sense of *taste* is often perverted, and the child constantly complains that his food is foul or has a metallic taste. This is especially the case when the buccal cavity is affected with stomatitis, or when rapid decay of the teeth has taken place. The breath acquires a sour and offensive odour, which is partly due to acid fermentation of the food, and partly to the liberation of fatty acids in the stomach. *Salivation* is frequently present, and the buccal secretions are usually thick and contain an excess of mucus. The *tongue* varies in appearance at different times of the day, being uniformly covered in the early morning with a thick white fur, which clears off toward mid-day, so that at night-time the anterior two-thirds of the organ often present a clean and moist appearance. At first the *bowels* are confined; but sooner or later the mucous membrane of the intestine becomes irritated and inflamed, and diarrhœa sets in. The secretion of *urine* is diminished, and the fluid deposits urates or crystals of uric acid upon standing. The surface of the body is cold and dry, but the hands and feet are apt to perspire profusely, and to emit an unpleasant smell. The *pulse* is small and feeble, and the temperature of the body subnormal. As long as the motorial functions of the stomach remain unaffected, and the processes of intestinal digestion unimpaired, the gastric disorder does not usually give rise to loss of flesh; but as soon as the food begins to stagnate in the stomach, emaciation and anæmia set in. The child then exhibits a thin, pale face, with dark areolæ around the eyes. The bowels are usually confined, and the stools are pale, and mixed with a large excess of mucus. From time to time diarrhœa occurs and persists for several days, during which time the motions are either pasty or liquid, and extremely offensive. In some cases, and especially in those where the disease has followed immediately upon an attack of measles or whooping-cough, several stools consisting almost entirely of mucus are passed every day. The child complains of constant headache, and is restless and irritable during the day, while at night its sleep is disturbed by dreams, or 'night terrors.' At this stage of the disease the tongue is usually red and glazed, and the mucous membrane of the mouth is apt to be attacked with aphthous ulceration. The patient grows gradually more emaciated and feeble, and the thinly-covered bones of his body and limbs present a marked contrast to his large and tumid belly.

From time to time subacute attacks of gastric or intestinal catarrh supervene upon the chronic complaint, and give rise to nausea, vomiting, and diarrhœa. The pulse is accelerated, the tongue thickly furred, and the temperature of the body becomes elevated and markedly intermittent. Each attack lasts several days, and tends still further to weaken the digestive powers of the stomach, and to increase the general condition of debility.

THE CHEMISTRY OF DIGESTION.

When the stomach is examined in the early morning, it is usually found to contain a small quantity of thick mucus which is slightly alkaline in reaction. Under the microscope columnar or polygonal epithelial cells, sarcinæ, bacteria and food débris may be observed. If the organ is evacuated after a test-meal, the food appears to have undergone but little digestion, and is largely mixed with mucus. Free hydrochloric acid is invariably absent, and the total acidity of the mixture is much diminished. The secretion both of pepsin and rennet is deficient, and the fluid, even after acidification, exerts but slight digestive action upon albumin. In most cases lactic acid is present in excess, and occasionally traces of butyric or acetic acid can also be demonstrated. With the progress of the disease, the quantity of the combined acid steadily diminishes, but it is only in those rare cases where atrophy of the gastric mucous membrane occurs that the acid entirely disappears. Even in the most severe cases it is usually possible to detect the presence of rennet, so that it would seem that this ingredient of the gastric secretion is the last to disappear in chronic inflammation of the stomach. In all cases, the motor power of the organ fails, so that the food stagnates and undergoes fermentation; and in many cases a considerable quantity can be extracted by the tube four or five hours after the last meal.

DIAGNOSIS.

As a rule, chronic gastro-intestinal catarrh can be easily recognised, the flatulent distension of the abdomen after meals, with pyrosis, nausea, and occasional vomiting being all symptoms which point strongly to the chronic affection of the digestive organs. Sometimes the mesenteric glands become enlarged and tuberculous as a result of the intestinal disease, but when this occurs careful palpation of the abdomen will usually reveal the presence of the caseous masses. The intermittent fever which is apt to accompany intercurrent attacks of subacute intestinal catarrh may occasionally suggest the presence of typhoid or tuberculosis, but in most instances the previous history of the case, combined with the absence of the other phenomena which accompany these latter complaints, is sufficient to lead to a correct diagnosis.

TREATMENT.

General.— In all cases the teeth should receive special attention, and such of them as are found to be diseased should be removed. The child must be made to brush the teeth two or three times a day with a suitable dentifrice, and also to wash out the mouth after each meal. When these precautions are adopted, it is often found that the putrid smell disappears from the breath, while the tongue assumes a more healthy appearance, and the appetite improves.

In healthy children, about three hours are usually required for the due performance of gastric digestion, but in cases of chronic catarrh of the stomach the process is much slower, owing to enfeeblement of the muscular coat of the organ. In order, therefore, to avoid maintaining the stomach in a state of perpetual functional activity, the frequency and size of the meals require careful regulation.

As a rule, three meals are sufficient during the course of the day, but sometimes it is expedient to allow a small quantity of light food during the evening. All excess of fluid with the meals must be avoided, but it is often useful to permit a tumblerful of hot water to be taken about an hour after each of the principal repasts. Alcohol is seldom necessary, and the routine habit of administering spirit-and-water, or wine, to a child suffering from dyspeptic symptoms is strongly to be deprecated. If a stimulant appears to be necessary, a little good claret well diluted with Vichy water may be allowed once a day. Excess of farinaceous foods must be prohibited, owing to the readiness with which they undergo fermentation, and it is therefore necessary to restrict the use of potatoes, bread, and other starchy articles of diet. Each mouthful of food should be carefully masticated before being swallowed, and the child should be taught to eat slowly, and to refrain from active exercise immediately after meals. If a subacute attack of gastric catarrh supervenes upon the chronic disorder, the diet must consist entirely of liquids until the urgent symptoms have subsided.

SCHEME OF DIET IN CHRONIC GASTRO-INTESTINAL CATARRH.

The best diet for your child is :

For Breakfast at 7.30 *to* 8 *a.m.*—Milk, peptonized or mixed with a small quantity of Vichy water ; cocoa made from the nibs, well diluted with milk ; thin toast or stale bread, with bacon fat ; a lightly-boiled egg ; a basin of bread-and-milk.

Avoid : Tea, coffee, sweet cocoas, excess of sugar and butter, hard-boiled eggs, jams, porridge.

For Dinner at 1 *p.m.*—A small quantity of strong soup, a piece of boiled sole, plaice, or whiting, a little stewed celery, asparagus, or French beans, and a milk pudding ; portion of a lightly-broiled mutton chop finely minced with gravy, lightly-cooked steak, or raw meat pulp, followed by plain jelly or a baked apple. Half a tumblerful of hot water may be taken at the end of the meal.

Avoid : Thick and strongly-seasoned soups, mackerel, boiled and salt meats, hashes and meats twice cooked, pork, veal, raw vegetables and fruits, much potato, beans, tomatoes, pastry, condiments, pickles, nuts, malt liquors, wines, cheese.

For Tea at 5 *p.m.*—Milk, cocoa made from the nibs, stale bread-and-butter, toast, rusks, a lightly-boiled egg, bread-and-milk, or a milk-pudding.

General Rules.

1. A tepid sponge or shower bath may be given each morning.
2. The under clothing should be of flannel, and a bandage may be worn round the waist during the day.
3. Active exercise should be taken in the open air each day.

Medicinal.—In all cases the action of the bowels must be regulated by means of some saline aperient, such as sulphate of magnesia, phosphate of sodium, Carlsbad salts, or by one of the natural purgative waters, like Hunyadi Janos or Friedrichshall. If the disease has arisen from the administration of unsuitable articles of diet, a few grains of blue pill or of gray powder, or a pill containing podophyllin, aloes and rhubarb, may be given once or twice a week with ntage. The carbonate and subnitrate of bismuth efly of value in those cases where irritability of mach is a prominent symptom. When nausea erienced after meals, a mixture containing carbo- of sodium, infusion of rhubarb, and infusion of an, given about half an hour before the food, usually allay it. Arsenic is of great value in the ment of obstinate cases attended by loss of appetite. As a rule, the acid preparation is the most suitable, and may be combined with dilute hydrochloric acid, and administered after meals. The dose should be gradually increased until the patient is able to take about twenty minims in the course of the day. Occasionally the oxide of silver, in doses of one-fifteenth to one-twentieth of a grain in the form of a pill, proves of value when everything else has failed, but the drug should never be continued for more than a week at a time. If abdominal distension occurs towards evening, an antiseptic such as resorcine, carbolic acid, or salicylic acid combined with pepsin and administered immediately after meals, will usually relieve this troublesome symptom. Lavage of the stomach is seldom required, but should it be deemed necessary, the organ may be washed out with warm water once or twice a week before breakfast.

CHAPTER VIII.

WEAK DIGESTION IN CHILDREN.

THE term 'weak digestion' is applied to a functional disorder of the digestive organs characterized by failure of the secretory and motor powers of the stomach and intestine.

ETIOLOGY.

In some instances a weak habit of digestion appears as a primary complaint; but in the vast majority of cases it ensues from damage inflicted upon the mucous membrane of the stomach by an attack of catarrhal inflammation.

In nearly 34 per cent. of the cases of primary failure of gastric digestion in children which have come under my notice, either one of the parents or another child in the family had suffered in a similar manner. The tendency to the complaint appears to be more often transmitted through the mother than the father, and, according to my experience, the disorder is more common in female than in male children. The only child of a family is particularly apt to fall a victim to the disease, and the same tendency is exhibited by the offspring of women who have married late in life.

Habitual constipation is one of the most frequent causes of feeble digestion. The atonic condition of the muscular coat of the colon causes the large bowel to perform its functions in a slow and imperfect manner, with the result that the stagnant food decomposes in the stomach, and gives rise to dilatation of its cavity. A neglected hernia in early life often induces a weak state of the digestive organs in a similar manner, and I have known cases of obstinate dyspepsia undergo rapid improvement as soon as measures were taken to keep the bowel within the abdominal cavity.

Another cause of weak digestion, which operates chiefly among the wealthy classes of society, consists in the persistent administration of peptonized foods. After an attack of gastric catarrh has passed away, some over-anxious mothers will continue to peptonize the major portion of the food for the child, under the impression that the disease will recur as soon as an ordinary diet is adopted. The result is that the stomach soon becomes accustomed to have its duties performed for it, and ceases to produce its natural secretion.

Among the lower classes, the converse of this is more often observed; and the child is either permitted to indulge in indigestible substances at all times in the day, or is regaled at irregular intervals with an amount of food far in excess of its powers of digestion. The stomach consequently becomes overworked, and chronic dyspepsia, with dilatation of the organ, is the usual result. The immoderate use of strong tea amongst the children of the poor is another cause of the feeble digestive powers so often encountered in hospital patients; while occasionally the immoderate use of tobacco in young boys appears to be responsible for the functional failure of the stomach.

In addition to these various conditions which exert an immediate influence upon the organs of digestion, there are others which operate in a less direct manner by inducing a deterioration of the general health. Among these must be reckoned insufficient muscular exercise, overcrowding, deficient ventilation, anæmia, and septic states of the mouth, arising from neglect of the teeth.[1] To these must be added another factor of no mean importance in the production of all functional disorders of the stomach in late childhood — namely, masturbation. This pernicious habit is extremely common in boys belonging to all classes of society, and may be initiated at a very early age, either from local irritation of the genitals by phimosis, or by personal example. The habit is difficult to cure, and when indulged in to excess is often followed by severe and intractable dyspepsia.

Occasionally the disorder appears to be connected in an intimate manner with the presence of intestinal worms, for as soon as the parasites have been destroyed the gastric symptoms rapidly subside. In female children a neglected leucorrhœal discharge is sometimes responsible for the digestive disturbance. Lastly, it may be noticed that children who have been born in India or certain parts of America, and afterwards come to reside in England, are extremely apt to suffer from a functional failure of the stomach, with recurrent attacks of subacute gastric catarrh.

---

[1] Several interesting examples of this have been recorded by my colleague, Mr. Denison Pedley, in his monograph on 'Diseases of Children's Teeth,' p. 159.

In 73 per cent. of my cases, the symptoms of weak digestion had either followed immediately upon an attack of acute inflammation of the mucous membrane of the stomach, or had gradually developed from the subacute form of the complaint. Some of the most severe and intractable cases are those in which the first signs of failure of digestion appear during the convalescent period of measles, typhoid, scarlatina, or influenza; and since parenchymatous gastritis is very apt to accompany the onset of these various infectious diseases, the subsequent disorder of the stomach is probably the result of partial destruction of the gastric glands (p. 223).

SYMPTOMS.

The process of food digestion is seldom accompanied by actual pain. More commonly a feeling of *weight* and *oppression* is complained of, or the child experiences a sense of suffocation shortly after the midday and evening meals. In some cases the skin over the centre of the sternum or pectoral regions feels bruised and tender to the touch, or a similar sensation is experienced between the shoulders in the region of the eighth and ninth dorsal vertebræ. In other instances, a kind of girdle pain is felt round the lower part of the chest, which appears to prevent full inspiration, and the child may be observed to constantly throw back its shoulders and straighten the spine with a view of overcoming this feeling of constriction. In other cases, again, abdominal distension is the main source of complaint, or the patient likens his sensations to those which he imagines would arise from the impaction of an iron bar across the lower end of the œsophagus. Occasionally a dull, gnawing sensation is felt in the epigastric region when the stomach is empty, which I have known described by many children as due to 'a hole in the stomach.'

These abnormal sensations usually develop within half an hour of a meal, and are capable of being temporarily relieved by the ingestion of stimulants and hot liquids. *Flatulence* is a common symptom, and is apt to give rise to considerable distress, especially towards evening. *Eructations* of gas occur after every meal, whatever be the nature of the food, and may be excited by drinking a little beef-tea, or even water. Flatulent distension of the intestines gives rise to attacks of colicky pain, accompanied by loud borborygmi. *Acidity* is seldom encountered, but occasionally a small quantity of rancid material may regurgitate into the mouth at the latter end of digestion. When acid eructations become continuous or severe, they usually denote the advent of subacute gastric catarrh. *Nausea* is a frequent symptom of the complaint, and usually makes its appearance soon after a meal. In severe cases, the feeling of sickness may persist all day, though capable of being temporarily relieved by the ingestion of food and stimulants. Under ordinary circumstances *vomiting* seldom occurs. In some children, however, the administration of fat or of some indigestible material is at once followed by emesis; and in certain cases the child will invariably vomit fish or meat within two hours of its ingestion. This form of gastric intolerance is chiefly encountered in those cases where weak digestion follows an attack of scarlatina or measles, but it may also be observed in children who have long been fed with peptonized foods. In some cases an attack of *hiccoughs* occurs regularly after the mid-day meal, and may persist for an hour or more.

The *appetite* is usually impaired, especially in the early morning, and the child may express the utmost loathing for food, and deliberately spit out everything which is placed in its mouth. More commonly the appetite is capricious, at one time being ravenous, and causing the child to devour any kind of food that may be offered to it, while at another the desire for food suddenly vanishes after a mouthful or two has been swallowed. Thirst is never a marked symptom of the disorder, and, indeed, the administration of liquid nourishment often appears to aggravate the symptoms. It may often be noticed, however, that the patient displays an inordinate affection for acid or sour substances, and will devour lemons or drink vinegar with avidity.

Constipation is always a prominent symptom of weak digestion. The stools are usually pale in colour, hard and dry, and occasionally consist of a number of small round balls, very similar in appearance to the excreta of a rabbit. In other cases the evacuations are pultaceous or frothy, while not infrequently the passage of undigested muscle fibre derived from the food gives rise to the erroneous supposition that the child is suffering from worms. The putrefaction of the intestinal contents is accompanied by the formation of gas, which accumulates in the cæcum and flexures of the colon, and produces *phantom tumours* in these regions of the abdomen.

The *tongue* is broad, pale, flabby, and indented along its margins by the teeth. In certain cases, and more especially in those where catarrh of the stomach is apt to supervene upon exposure to cold, the tongue often exhibits a form of superficial ulceration, the characters of which have already been described (p. 232). In all cases the *pulse* is weak and compressible, and its rhythm easily disturbed by excitement or by the ingestion of food. Occasionally I have observed the pulse rate to fall as low as forty or fifty beats per minute during the early period of digestion. *Palpitation* is frequently complained of, and may be accompanied by a sense of *giddiness* or *faintness.*

Although actual emaciation does not usually accompany this condition of deficient digestive power, the child does not thrive. On the contrary, it remains pale, thin and languid, at one moment exhibiting an extreme degree of vivacity, at the next becoming dull and spiritless, and disinclined to the slightest mental or physical exertion. The *skin* loses its natural elasticity, and becomes soft and clammy, while sudden and profuse perspirations are apt to occur after meals or during the early part of the night. The *urine* is diminished in amount, neutral or faintly acid in reaction, and often deposits a quantity of phosphates upon standing. Sometimes it is opaque when voided, and the patient is then apt to complain of soreness or pain along the course of the urethra or at the orifice of the meatus.

In addition to these general symptoms, which are common to all cases of weak digestion, there are several others of a more special nature which deserve attention.

Many dyspeptic children suffer from extreme *languor* and *drowsiness* after the principal meal in the day, and not infrequently fall into a short sleep from which they awaken irritable and unrefreshed. Occasionally loss of memory or a strange confusion of ideas may be observed during the course of the afternoon, or the child incessantly nods its head, blinks the eyes, or indulges in other *choreic movements.* In certain cases cardiac failure occurs as the result of over-distension of the stomach with gas or food.

After a period of unusual vivacity or excitement, the patient suddenly complains of giddiness, sickness or faintness, becomes pale and cold, and, when placed upon the bed, may lie for a considerable time in a condition of semi-unconsciousness. The eyes are half closed and devoid of expression, while dark lines gather round the mouth. The pulse is slow, feeble, and sometimes hardly perceptible, and a cold sweat appears on the forehead and neck. As a rule, the attack terminates in the expulsion of a large quantity of gas from the stomach, or by a copious evacuation of the bowels.

In some children, about the age of five or six, I have observed *cutaneous phenomena* which were apparently due to a disturbance of the vaso-motor system. One of the most curious of these consists of a sensation of extreme heat and dryness of the palms of the hands and soles of the feet. In such cases it is not uncommon to see the little patient repeatedly spit upon the hands and wave them in the air with the idea of cooling them, and I have been several times informed that the nurse was obliged to get up in the night and sponge the child's hands and feet with water in order to relieve this troublesome symptom. Another phenomenon which occurs in older children is the appearance of dull red patches upon the hypothenar or thenar eminences during the process of digestion. On examination of the affected parts, a distinct subcuticular purple flush can be seen, which disappears on pressure, and is accompanied by a sensation of intense burning or itching. This particular symptom is at times replaced by hyperæsthesia over the distribution of the radial nerve, or by a sense of numbness and tingling in the fingers. Flushing of the face or burning of the ears and nose after meals is of frequent occurrence, and the slightest excitement is apt to determine a large quantity of blood to the head and neck, and to produce dimness of vision and noises in the ears. Occasionally small patches of erythema make their appearance upon the forehead, face and neck, and persist for several hours. The subjects of feeble digestion are also liable to attacks of urticaria after eating certain articles of food, the ingestion of which under ordinary circumstances produces no bad effects; among these may be mentioned fish, porridge, almonds and strawberries. Occasionally an attack of *pseudo-asthma* occurs within a few hours of a meal, and is accompanied by extreme dyspnœa and cyanosis (p. 230). These symptoms rapidly subside after emesis has taken place. Another effect of imperfect digestion upon the respiratory centre is to be found in the constant *yawning* and *sighing* which is observed in many cases after meals, and in the short, dry *cough* which occasionally follows the administration of indigestible forms of food.

CHEMISTRY OF DIGESTION.

When the contents of the stomach are examined after a test-meal, the particles of bread appear to have undergone but little digestion. The mixture filters easily owing to an absence of mucus, and is acid in reaction, but the total acidity is diminished. Free hydrochloric acid can usually be recognised, but on quantitative examination both it and the combined acid are found to be considerably diminished. In many cases the presence of lactic acid can be demonstrated. The filtrate digests albumin feebly, even after acidification, but if dilute hydrochloric acid is introduced into the stomach and withdrawn after a short time, the fluid is found to possess powerful digestive properties. The motor power of the stomach, as shown by the stagnation of the food and failure of the salol test, is markedly feeble, and the organ can be easily ballooned when air is pumped into it through a hand-bellows. From these facts, it would appear that in cases of weak digestion the main symptoms of the complaint arise from failure of the secretory and motor functions of the stomach.

TREATMENT.

General.—The general principles of treatment are the same as those which are observed in cases of atonic dyspepsia in later life. Regular exercise in the open air is matter of the utmost importance, and should be indulged in both summer and winter when the weather is fine. The child should never be permitted to sleep in its nursery, but should be provided with a cot in another room. The clothing must be warm, and during the cold months of the year the underclothes should be made of flannel or wool, and completely cover the arms and thighs. At night-time a flannel pyjama suit is to be preferred to a loose cotton garment, owing to the profuse perspirations which are apt to occur.

The skin should be maintained in a healthy condition by the employment of a sponge bath each morning, to which, if a more stimulating effect is necessary, a moderate quantity of brine or sea-salt may be added. Owing to the feeble state of the circulation, the employment of cold water is apt to produce unpleasant symptoms, so that it is wise to use tepid baths at first, and to gradually lower the temperature of the water as the child becomes accustomed to the treatment, and its general health improves.

In chronic cases a change of air is often of considerable benefit, but whenever there is a tendency to gastric catarrh, the child should be sent to some bracing inland health resort rather than to the seaside.

Diet.—One of the most difficult problems in the treatment of weak digestion in children is the selection of suitable food. [illegible] cases of [illegible] milk [illegible] forms of liquid food [illegible]. Fluid nourishment [illegible] the stomach is inflamed, [illegible] abnormally irritable condition [illegible] mischief present [illegible] the administration of [illegible] quantities of fluid only [illegible] render it still [illegible]. Another [illegible] deserves notice is [illegible]. The stomach is [illegible] difficult to correct, and to a great extent every individual is responsible for the manner in which his stomach performs its functions. Thus, if a person habitually indulges in an excess of nitrogenous food, the organ becomes accustomed to produce an abnormal amount of acid juice at each meal, and the symptoms of acid dyspepsia are apt to develop. On the other hand, if the stomach is constantly supplied with materials which have already undergone peptonization, the normal stimulus to secretion that is afforded by the introduction of food is lost, and the digestive functions soon become impaired through lack of work.

Alimentary substances vary considerably as regards the facility with which they are dissolved by the gastric juice, and in cases where this is only secreted in an imperfect manner, the relative digestibility of the different kinds of food becomes a matter of the greatest moment. As regards animal food, the more tender the fibre, the more easily is it dissolved in the stomach ; hence mutton is more digestible than beef, and the latter than veal or pork. Chicken, pigeon, and the smaller kinds of game, offer less trouble to a feeble digestion than the flesh of the turkey, goose, and duck. The larger forms of fish are less easy of digestion than those whose tissues are softer ; hence, while sole, haddock, whiting and plaice are extremely serviceable in cases of weak digestion, cod, mackerel, herring and salmon must be excluded from the dietary on account of the difficulty they present to solution by the gastric juice. The fat portions of meat are very prone to undergo decomposition in a stomach where the secretion of hydrochloric acid is deficient, and should therefore be avoided as far as possible. It is also worthy of notice that the digestibility of the various forms of food depends, to a great extent, upon the manner in which they are cooked, such processes as stewing and boiling tending to produce swelling and separation of the individual fibres, and thus to permit of their permeation by the gastric juice.

As a rule, green vegetables should figure but slightly in the dietary of children suffering from weak digestion, owing to the large proportion of indigestible material they contain. Those which possess a large amount of starch are also contra-indicated in the early stages of the complaint, when flatulence is apt to follow the ingestion of food. Fruit digests more readily after being cooked, and stewed apples, pears, or prunes, form a valuable addition to the meals when the stomach has commenced to recover its normal tone. Fresh and stony forms of fruit can seldom be taken without discomfort, while those which have been dried by exposure to the sun are extremely indigestible, and must therefore be prohibited.

Stale bread is more readily dissolved in the stomach than that recently cooked, and the aerated variety is more easily digested than the coarser and heavier kinds. In all cases the child should be obliged to spend an adequate time at the table, and the habit of bolting one part of the meal with the view of hastening the arrival of a later and more favourite dish must be discouraged. At the commencement of the treatment the food should be carefully minced before being administered, and the child must always be made to chew each mouthful well before swallowing it. Sometimes a decayed or painful state of the teeth prevents proper mastication of the food, and in such cases the condition of the mouth must receive special attention.

Only a small quantity of fluid should be taken with the meals, and this should be sipped at intervals rather than swallowed in bulk. Effervescing waters usually increase the tendency to flatulence, and are therefore unsuitable in the majority of cases. Milk usually agrees when diluted with water, but when taken in the pure state it is often digested with difficulty, and is apt to give rise to pain in the abdomen, and constipation. The sweet forms of cocoa also produce symptoms of an unpleasant nature, owing to their tendency to undergo fermentation; but the decoction of the cocoa-nibs is free from this objection, and forms an agreeable drink when mixed with milk. Tea and coffee are always injurious at first, but when the disorder improves, a little weak tea containing milk often affords a pleasant change. Alcoholic liquors are seldom necessary, but in severe cases a little good brandy or whisky, diluted with warm water and given at the end of the meal, proves a valuable remedy. Stout and porter are apt to give rise to acidity and nausea, and should be avoided.

SCHEME OF DIET IN WEAK DIGESTION.

The best diet for your child is:

For Breakfast at 8 *a.m.*—Milk, diluted with one-third of water; cocoa from the nibs, weak tea with milk, bread-and-milk, thin toast, stale bread-and-butter, a lightly boiled egg, a slice of bacon or tongue.

Avoid: Coffee, strong tea, newly-baked bread, salt or dried fish, hard-boiled eggs, pastry, jams.

For Dinner at 12.30 *to* 1 *p.m.*—Grilled mutton chop, roast mutton or lamb, fried sole or plaice, rabbit, roast chicken, pigeon, tripe without onion, good potato, custard, semolina or sago pudding, stale bread with a little butter, stewed fruit, milk.

Avoid: Salt meat or meat twice cooked, soups, broths, veal, pork, mackerel, herring, green vegetables, pastry, cheese, sauces and pickles, malt liquors.

For Tea at 5 *p.m.*—Milk or weak tea, cocoa from the nibs, stale bread or thin toast with a little butter, a lightly-boiled egg, bread-and-milk, custard or tapioca puddings.

General Rules.

1. Excess of fluid with the meals must be avoided. Half a tumblerful of hot water may be sipped at intervals.
2. Each mouthful of food must be thoroughly masticated before being swallowed, and meat or fish minced finely upon the plate.
3. No food should be allowed between the meals, except where it seems advisable to permit a cup of warm milk at 11 a.m. or 8 p.m.
4. Daily exercise in the open air should be taken whenever the weather is fine, but fatigue immediately after the meals must be avoided.
5. The clothing should be warm. A tepid salt bath may be administered every morning.

Medicinal.—It is the custom to regard the condition of feeble digestion as an indication for the use of tonic remedies, and for this purpose iron, quinine, and nux vomica are usually administered with some bitter infusion. As a result of this treatment, the patient usually loses the last vestige of appetite he possessed, and either suffers from a constant feeling of nausea or develops an attack of gastric catarrh. The only successful method of treating these troublesome cases is to abandon all attempts to produce immediate and striking results, and to endeavour to aid the muscular contraction of the stomach by preventing stagnation and fermentation of the food.

Children who suffer from constipation often exhibit a loose state of the bowels, which gives rise to the belief that they are suffering from diarrhœa. An examination of the stools, however, is usually sufficient to prevent a mistake of this kind, since the dejecta are found to be small in quantity, and to consist merely of a little fluid containing a few lumps of hard fæcal material. In such cases, it is not unusual to find that the rectum and the transverse or descending colon are blocked by solid masses of fæces. Whenever, therefore, constipation exists, and more especially if the child has been confined for some time to a milk diet, it is wise to commence the treatment with a full dose of castor-oil, and to repeat it each day until the stools have assumed a normal appearance. In some cases relief can only be procured by the administration of enemata of soap-and-water or castor-oil, or by forcibly evacuating the contents of the rectum with the finger or a spoon. When castor-oil produces vomiting, a saline aperient may be used; but severe and drastic purges must be avoided until all signs of mechanical obstruction have passed away. The relief afforded by evacuation of the bowels makes itself apparent at once, and I have known more than one case in which the phenomena of feeble digestion completely disappeared as soon as the bowels had been cleared out and solid food substituted for a milk diet.

Functional failure of the stomach and intestine is usually associated with a deficient elimination of bile, so that if the stools are pale in colour and offensive, or if the child suffers from nausea in the early morning, a small dose of gray powder, calomel, or blue pill should be given on alternate evenings for a week, followed the next day by a saline draught. As soon as a daily evacuation of the bowels has been secured, an attempt should be made to inhibit the process of food fermentation by the administration of suitable antiseptics. For this purpose carbolic acid is the most reliable, and can be advantageously given in the form of the glycerine preparation (B.P.), of which from four to six minims may be administered in chloroform-water two or three times a day. Young children sometimes dislike the taste of carbolic acid, so that it may be necessary to employ resorcine (four grains) instead. In either case the antiseptic mixture should be given about an hour and a half after meals. Within ten days or a fortnight the symptom of abdominal distension has usually subsided, and measures can then be taken to stimulate the secretion of gastric juice. As a rule, an alkali combined with rhubarb and infusion of gentian is of great value at this period of the complaint, and may be administered about an hour before the two principal meals of the day. In older children, or in cases of a very chronic character, pepsin with hydrochloric acid after meals is usually more efficacious, either the liquor pepticus (Benger) or the acid glycerine of pepsin (Bullock) being employed for the purpose. Occasionally a pill containing pepsin, ipecacuanha, ginger, capsicum, and gentian is of considerable service when given immediately after meals.

It is only when the principal symptoms of the disorder have disappeared, and the bowels have been properly regulated, that tonic treatment can be commenced with safety. The bland preparations of iron, such as the tartrate or the dialysed solution, are the most suitable, and may be given with one or two drachms of glycerine twice a day after meals. In some anæmic children, one grain of the dried sulphate of iron, combined with half a grain of aloin and two grains of pepsin, makes an excellent pill for administration after food; but in all cases where iron is employed, a dose of mercury must be given once or twice a week. Quinine and arsenic seldom agree. Cod-liver-oil is a valuable remedy in some cases, and may be advantageously given either along with the solution of tartrate of iron or combined with extract of malt.

CHAPTER IX.

ULCERATION OF THE STOMACH

(HÆMATEMESIS—MELÆNA—STENOSIS OF THE PYLORUS).

SEVERAL varieties of ulceration of the stomach are encountered during the period of infancy and childhood. In some cases the disease arises from acute catarrh of the mucous membrane, in others a small interstitial hæmorrhage is the immediate cause of a localized necrosis ; while in others, again, inflammation of the solitary lymphatic glands, or the deposition of tubercle, is responsible for the process of ulceration. All authorities are agreed, however, that a simple perforating ulcer similar to that which occurs during adult life is extremely rare before puberty. Thus, among the 252 cases of gastric ulcer collected by Lebert,[1] only one occurred before the age of ten, and Welch[2] only found one instance in the first decade of life among the 607 cases which he investigated. Brinton[3] includes two similar instances in his series of 226 cases of gastric ulcer, while Rokitansky,[4] in the whole of his wide experience, never encountered the disease in a child under fourteen years of age. Although it has been possible to collect from the literature a considerable number of cases which purport to be examples of simple gastric ulcer, careful examination shows that in the great majority the disease of the stomach ensued as the result of some other complaint, and was not, therefore, pathologically identical with the idiopathic form which is encountered in later life. In the following pages a short description will be offered of the different varieties of gastric ulceration which are occasionally observed in infancy and childhood, namely: (1) hæmorrhagic erosions; (2) follicular; (3) simple, acute and chronic; (4) tubercular; (5) malignant. To this will be added a few remarks upon the subject of congenital stenosis of the pylorus.

---

[1] 'Path. Anatom.,' p. 199. [2] Pepper's 'System of Medicine,' vol. ii., p. 483.

[3] 'Ulcer of the Stomach,' 1857, p. 9.

[4] Cited by Rehn, *Jahrb. f. Kinderheilk.*, 1874, s. 19.

1. HÆMORRHAGIC EROSIONS.

Small extravasations of blood into the mucous membrane of the stomach are frequently observed after death. In many cases the hæmorrhage appears to have arisen from congestion of the gastric capillaries, consequent upon failure of the circulation. Thus, dilatation of the heart from endocarditis, chronic pneumonia, or tuberculosis is often accompanied by punctiform extravasations into the tissue of the stomach; while in rarer instances cirrhosis of the liver or other causes of portal obstruction are responsible for the rupture of minute vessels in the inner coat of the organ. Discrete hæmorrhages in the gastric mucous membrane are also apt to occur during the course of such diseases as purpura, scurvy, hæmophilia, icterus, and leucocythemia, as well as in all inflammatory diseases of the stomach itself. The size of the hæmorrhage varies considerably, being sometimes so minute as to require the microscope for its detection, while in other cases areas of the mucous membrane several millimetres in diameter are densely infiltrated with blood. As a rule, the disease presents a circular or oval shape, but occasionally it occurs in the form of linear or flame-shaped streaks. With regard to the site of the hæmorrhages, it may be observed that when they arise from chronic congestion of the stomach they usually occupy the fundus of the organ in the immediate neighbourhood of the great curvature; while in those cases where catarrh of the mucous membrane is the immediate cause of the mischief, the extravasations are most numerous in the pyloric region, near the lesser curvature. When of recent origin, the disease exhibits a vivid red colour; but after it has existed for a few hours it acquires a brownish or black tint, owing to the action of the acid gastric juice upon the colouring matter of the blood. In most cases the hæmorrhagic effusion is strictly limited to the mucous coat of the stomach, but occasionally the whole thickness of the wall may be infiltrated with blood.

Whenever the hæmorrhage extends for any depth into the mucous membrane, the affected tissue either necroses and becomes detached, or is dissolved out by the action of the gastric juice. The so-called 'erosions' which are thus produced are slightly oval or circular in outline, and are surrounded by an elevated and tumid ring of a pale yellow colour. The base of the ulcer is most commonly situated in the deeper portions of the mucous membrane, but if the antecedent hæmor- ige has been extensive, the disease may involve the scular, or even the serous, coat of the organ.

Under the microscope the hæmorrhagic origin of the erosion is easily recognised by its ragged edge, and the presence of blood-corpuscles in the surrounding tissue. The natural tendency of this form of ulceration is to rapid and complete healing; but if the erosion is extensive and deep, or if there exists some local or constitutional obstacle to repair, the disease may assume a chronic and progressive character. In rare cases severe hæmorrhage results from the rupture of a small artery.

2. FOLLICULAR ULCERATION.

In many cases of pulmonary tuberculosis and other infective diseases,[1] the mucous membrane of the stomach presents numerous small circular ulcers, which vary in size from a few lines to two centimetres in diameter. The edges of these excavations are slightly raised, and either of a pale yellow colour, or surrounded by a zone of highly-injected capillaries. The disease seldom extends deeper than the submucous coat, but occasionally the mischief appears to be progressive, and the base of the ulcer may then involve the muscular, or even the serous, tissue. Here and there small hemispherical masses, about the size of a pin's head, may be observed upon the surface of the mucous membrane. These appearances are due to inflammation of the solitary lymphatic glands situated in the wall of the stomach, and it is from necrosis of these structures that the ulcers are produced (Fig. 17).

With the aid of the microscope the various stages of the disease can easily be discerned. At first the solitary gland becomes enlarged and swollen, and the mass tends to lose its definite outline. After a time many of its cells may be seen to encroach upon the neighbouring tissues, and to form a layer over the surface of the muscularis mucosæ for a considerable distance on either side of the lymphatic follicle (Fig. 18). The swollen gland presses upon the gastric tubules which surround and cover it in, and gradually causes destruction of those in its immediate vicinity. The mucous membrane over the surface of the follicle becomes thinned out and atrophied from pressure, and

---

[1] Billard and Cruveilhier described follicular ulceration as of common occurrence in new-born infants.

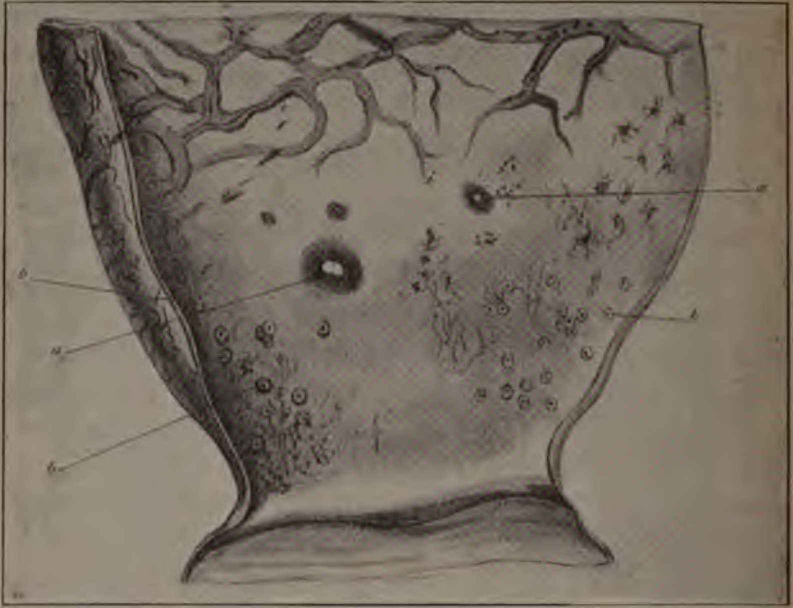

FIG. 17.—Drawing of the Pyloric End of the Stomach, showing Inflammation of the Solitary Glands, with Acute Ulceration originating in these Structures. (After Carswell.) *a*, Perforating ulcers; *b*, inflamed solitary glands.

in many instances a portion of it is drawn into the centre of the adenoid mass, and undergoes rapid disintegration (Fig. 19).

About the same time the cells in the centre of the follicle lose their individual outlines, and undergo a process of softening, so that in a short time the interior of the gland becomes semi-fluid in consistency. The final stage of the disease consists in rupture of the thin layer of tissue which covers the minute abscess,

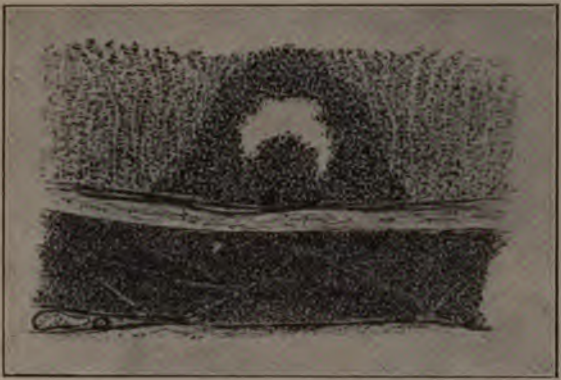

FIG. 18.—Section of a Stomach affected with Catarrhal Inflammation, showing an Enlarged Solitary Gland. (× 30.)

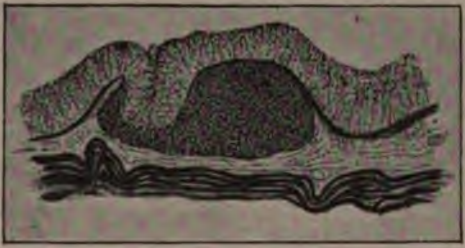

FIG. 19.—Section of a Stomach (Guinea-pig), showing an Enlarged Solitary Gland in the Submucous Tissue, with inversion of the Mucous Membrane. (× 40.)

the contents of which are discharged into the cavity of the stomach (Fig. 20).

The subsequent fate of the ulcer depends upon the activity of the gastric juice, the disease either healing or enlarging its circumference. This latter condition sometimes occurs in children who are suffering from enteric fever, and I have seen two cases in which severe hæmatemesis ensued from this cause. Fig. 21 represents a drawing of a stomach taken from a girl eight years of age who succumbed during the third week of enteric fever. Four well-defined ulcers were found in the pyloric region, one of which presented a loosely adherent slough. The edges of the ulcers were sharply defined and somewhat undermined, while their bases were situated in the submucous and muscular coats of the organ. On microscopic examination, the lymphoid tissue of the stomach was found to be enormously increased, and the supposition that the ulcers originated in disease of the solitary glands was confirmed by the appearances of the smallest one. From these facts it would appear that, under certain circumstances, disease of the solitary gastric glands may give rise to a form of perforating ulcer of the

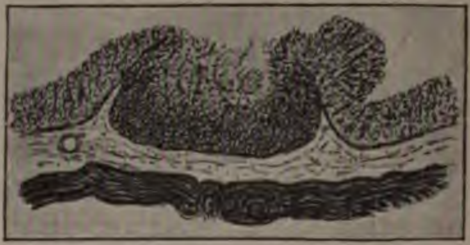

FIG. 20.—Section of a Stomach (Guinea-pig) affected with Follicular Ulceration. (× 40.)

stomach which closely resembles the idiopathic type of the disease.

3. SIMPLE ULCER.

In many cases the loss of substance presented by the mucous membrane is circular in shape, and so sharply defined that it appears as though it had been removed with a punch. The edges of the sore are perpendicular, smooth, and soft, and its base is situated in the muscular or serous coat of the stomach, while the surrounding tissue shows no signs of inflammatory

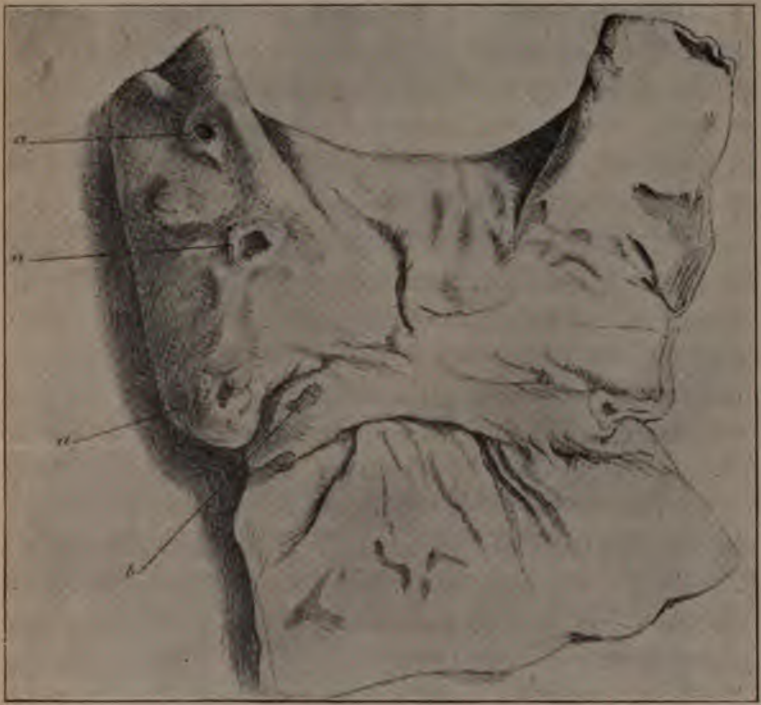

FIG. 21.—Drawing of the Pyloric End of the Stomach in a Case of Enteric Fever. *a*, Acute perforating ulcers with clean bases ; *b*, an ulcer with adherent slough.

thickening. In these cases the gastric symptoms appear suddenly, and hæmatemesis or perforation may be the first phenomenon to attract attention. In other instances the ulcer is of much larger size, and exhibits a sloping, 'terraced,' and indurated edge. The base of the sore is uneven, and not infrequently adherent to some neighbouring organ. From these differences in the general aspect of the disease, it appears to be justifiable to consider that a gastric ulcer may either be acute or chronic in character, and it is upon this basis of distinction that the following cases will be considered.

(*a*) *Acute Perforating Gastric Ulcer.*

1. Billard[1] relates a case of gastric ulcer in an infant fifteen days old. The disease was situated in the pyloric end of the stomach, in the neighbourhood of the great curvature. It was circular in form, and had penetrated to the peritoneum. The edges and base of the sore were dark brown in colour.

2. The same writer has recorded the case of an infant who died of hæmorrhage from the stomach and intestine five days after birth. At the necropsy, a circular ulcer the size of a gulden piece was found in the stomach near the cardiac orifice. The edges of the ulcer presented a lacerated and blackened appearance, 'as though they had been burnt.'

3. Busch[2] records the results of a post-mortem examination upon a male infant seven days old, where an ulcer the size of a pfennig piece was found on the anterior wall of the stomach, near the great curvature, and close to the pylorus. The edges of the ulcer

---

[1] 'Krankheiten d. Neugebornen u. Säuglingen,' 1829.

[2] *Hufeland's Journal*, 1836, s. 123.

were ragged, and the disease had exposed the peritoneum.

4. Buzzard[1] relates the case of a girl aged nine years, who was suddenly attacked with violent pain in the belly, and shortly afterwards succumbed to general peritonitis. At the necropsy, a small funnel-shaped ulcer was found in the centre of the lesser curvature of the stomach, which had perforated all the coats of the viscus. The patient had suffered for a short time from discomfort after food.

5. Rehn[2] records the case of an infant twenty-one months old, who succumbed to general tuberculosis. After death two ulcers were found in the stomach. The larger was situated on the lesser curvature, about twenty-five millimetres from the cardiac orifice; while the smaller one, which was more superficial in character, occurred in the centre of the stomach, about four millimetres from the great curvature. There were no signs of tubercle.

6. Binz[3] observed an ulcer in the centre of the lesser curvature of the stomach in a female child eleven days old.

7. The case described by Goodhart[4] is one of considerable interest. The subject was an infant who died from hæmatemesis thirty hours after birth. On examination, the stomach was found distended with clot, and in the cardiac extremity of the organ, close to the great curvature, there was a small oval-shaped ulcer, measuring one-eighth of an inch across. The edges were raised and clearly defined, and the sur-

---

[1] *Path. Soc. Trans.*, vol. xii., p. 84.

[2] *Jahrb. f. Kinderheil.*, 1874, s. 19.

[3] *Berl. Klin. Woch.*, 1865, No. 15.

[4] *Path. Soc. Trans.*, 1881, vol. xxxii., p. 79.

rounding tissue was injected with blood. Upon the base of the ulcer there was a black speck, which proved on examination to be an eroded bloodvessel.

8. Barlow[1] relates the case of a female child, aged twenty-one months, who succumbed to general tuberculosis. On the anterior wall of the stomach, close to the great curvature, was an oval ulcer, which measured one-quarter of an inch by one-eighth of an inch. The edges were thin and undermined, and the loss of substance extended almost to the peritoneum. There was no appearance of induration. Two other ulcers existed in the middle zone of the organ, near the lower margin, and two more in the pyloric region. Near the last-named there was a small hemispherical elevation beneath the mucous membrane which resembled a tubercle.

9. Colgar[2] has recorded the case of a female child, two and a half years of age, who died of convulsions accompanied by severe pyrexia. At the post-mortem examination, an ulcer was found on the posterior wall of the stomach, near the lower border of the fundus, perforation of which had given rise to general peritonitis.

Among the records of the London Hospital for the last thirty years, I have found four cases where an acute ulcer was found in the stomach of a child less than fourteen years of age.

10. A girl, thirteen years old, was admitted into the hospital with the symptoms of typhoid fever of eight days' duration. Vomiting occurred once or twice, but there was no complaint of epigastric pain. At the end of the fourth week of the disease, when the tem-

---

[1] *Path. Soc. Trans.*, 1887, vol. xxxviii., p. 141.

[2] *Medical News*, 1892, p. 409.

perature had begun to decline, the patient was suddenly seized with severe hæmatemesis, after which she became unconscious and died. At the necropsy, the anterior wall of the stomach was found to be adherent to the under surface of the liver. Scattered over the inner surface of the stomach there were numerous sharply-defined ulcers, the largest of which was about the size of a florin. The edges were thin and undermined, and the base formed by the muscular or peritoneal coat. In the first part of the duodenum there was an ulcer of a similar character, while the whole of the intestine, from the jejunum to the rectum, was riddled with typical typhoid ulcers.

11. A girl, aged twelve years, was admitted into the hospital for severe burns of the arms and legs, from which she died at the end of a week. In the centre of the lesser curvature of the stomach there was a circular ulcer the size of a sixpence, evidently of recent formation. The mucous membrane of the organ was intensely injected, and presented several small ecchymoses. The duodenum also showed signs of inflammation, but no ulceration could be found.

12. A boy, aged ten years, was admitted suffering from acute pneumonia of the right side. At the necropsy the stomach was found distended with blood-clot. On the lesser curvature, one inch from the pylorus, there were two round ulcers, separated from one another by a thin bridge of mucous membrane. The larger one measured one inch, and the smaller about half an inch, in diameter. Both ulcers exhibited sharp edges and had penetrated to the peritoneum. On the floor of the larger ulcer there was an eroded vessel which admitted a fine probe.

13. A female child, twenty months old, succumbed in the hospital to noma following varicella. On the anterior wall of the stomach, near the pylorus and close to the lesser curvature, there were two small ulcers with well-defined edges.

(*b*) *Chronic Simple Ulcer.*

14. Reimer[1] relates the case of a girl, three and a half years old, who was admitted into hospital with the history of having suffered from indigestion for nearly two years. The patient improved under treatment, and was about to be discharged, when she contracted measles. During the pyrexial period of this disease she complained of severe pain in the region of the umbilicus, and vomited incessantly. A few days later hæmatemesis occurred, and the child rapidly sank, and died of exhaustion. At the necropsy the inner surface of the stomach was found to be injected with blood, and to present numerous punctiform hæmorrhages. On the posterior wall of the viscus, near the lesser curvature, and situated about two centimetres from the pylorus, there was a circular ulcer, the base of which was formed by the pancreas, to which it was bound by fibrous adhesions. Erosion of a branch of the pancreatic artery was the cause of the hæmorrhage during life.

15. Gunz[2] records the case of a boy, five years of age, who died of scarlatina after having suffered for several months from pain and vomiting after food. About one and a half inches from the pylorus, near the great curvature of the stomach, and on its posterior

---

[1] *Jahrb. f. Kinderheilk.*, 1876, s. 289.

[2] *Ibid.*, 1862, s. 161.

wall, there was found a simple ulcer the size of a gulden piece. The shape of the ulcer was elliptical, its long axis coinciding with that of the stomach, and its edges, though sharply defined, were markedly indurated. The base of the ulcer was adherent to the transverse colon. There were also evidences of chronic gastro-intestinal catarrh, accompanied by ulceration of the large bowel.

16. In a case published by Eross,[1] a chronic ulcer of the stomach was found to be associated with two others of more recent origin. The patient was a girl, twelve years old, who succumbed to an attack of general tuberculosis, during the progress of which disease she had complained of occasional pain and tenderness in the epigastric region. Nausea was present after meals, but vomiting and hæmatemesis were never observed. After death the stomach presented three ulcers on its posterior wall near the lesser curvature. Two of these were small in size, superficial in character, and obviously of recent formation. The third measured nearly two and a half centimetres in diameter, and exhibited distinct thickening of its edges. It had completely perforated the walls of the organ.

17. Rufz[2] reported the case of a girl, thirteen years of age, who, after suffering for several months from symptoms of moderate dyspepsia, was suddenly attacked a few hours after a meal with severe pain and vomiting, and died of peritonitis. At the necropsy a chronic ulcer, measuring two centimetres in diameter, was found in the stomach, perforation of which had caused death.

---

[1] *Jahrb. f. Kinderheilk.*, 1879, s. 331.

[2] *Gaz. Médic.*, 1843, p. 673.

18. Chvostek[1] has described a remarkable case in which the symptoms of gastric ulcer were present for fourteen years. A boy at the age of four was exposed to severe cold, and was immediately afterwards seized with nausea and discomfort at the epigastrium after food. The complaint varied in severity from time to time, but after the lapse of a few years it became firmly established, and pain ensued after each meal, and often lasted for many hours. At the age of eighteen, when he died, the patient was extremely emaciated, and resembled in stature and general appearance a boy of ten. At the necropsy the stomach was found to be enormously dilated. Near the cardiac orifice there was a round ulcer about three centimetres in diameter and half a centimetre in depth, the edges of which were clean-cut and devoid of thickening. Another ulcer was present close to the pylorus, on the posterior wall of the stomach, and measured two and a half centimetres in diameter. The edges of the sore were very thick and the surrounding tissue cirrhotic, while the base was formed by the head of the pancreas, to which the stomach was firmly adherent. The contraction of this ulcer had so narrowed the pyloric orifice that it would only admit the tip of the little finger. The whole of the muscular tissue of the stomach was hypertrophied.

19. Leith[2] has recently recorded the case of a girl, ten years of age, who died of empyema consequent upon the perforation of the diaphragm by a gastric ulcer. The child had enjoyed excellent health until she was attacked with acute pneumonia of the left

[1] *Jahrb. f. Kinderheilk.*, 1883, s. 364.

[2] *Internat. Clinics*, 1895, vol. iv., p. 49.

side. About a fortnight after the crisis the temperature began to rise and the child rapidly lost flesh. Shortly afterwards an œdematous swelling appeared in the second left intercostal space, which when incised gave exit to some fœtid pus. A few weeks later signs of a localized empyema were discovered in the left side of the chest, and an operation was performed. The patient, however, gradually grew worse, and died about eleven weeks after the onset of the pneumonia. At the autopsy acute pericarditis was found to have been the immediate cause of death. A small perforation in the left side of the diaphragm communicated by a sinuous track with a localized pyo-pneumo-thorax in the left chest. Just in front of the greater curvature of the stomach, and situated four and a half inches from the cardiac orifice, and about eight inches from the pylorus, there was an ulcer which measured one-quarter by three-sixteenths of an inch. The edges of the sore were not thickened, and the floor was formed of the peritoneal and muscular coats of the organ. The base was adherent to the spleen, and from it a suppurating track passed upwards through the hole in the diaphragm into the pleural cavity. Bacteriological examination of the pus gave an almost pure culture of the *Staphylococcus pyogenes aureus*.

ETIOLOGY.

An examination of the various cases of *acute* gastric ulcer which have been recorded helps to throw a certain amount of light upon the etiology of the disease. In four out of the five cases where the complaint developed immediately after birth, the ulcer was found to occupy the cardiac region of the stomach, close to the greater curvature. It has already been observed that this region of the stomach is the usual site for hæmorrhagic erosions resulting from congestion of the gastric vessels, and the fact that in two instances the edges of the ulcer exhibited a lacerated and blackened appearance affords strong support to the theory that an effusion of blood into the mucous membrane of the stomach was the cause of the tissue necrosis.

That such an accident should occasionally occur does not appear improbable, when it is remembered how frequently symptoms of asphyxia are observed in an infant after difficult delivery. When the erosion is small, it usually heals rapidly and produces no ill effects ; but if the effusion of blood affects a considerable area of the mucous membrane, the resultant ulcer may erode some large bloodvessel and give rise to hæmatemesis or melæna, as in the cases recorded by Billard and Goodhart.

In two cases the disease developed during the course of general tuberculosis, both the patients being less than two years of age. In neither instance did the ulcerative process in the stomach exhibit any evidence of a tuberculous origin, although in one case (Barlow) a suspicious growth was found in the neighbourhood. This association of gastric ulcer with pulmonary tuberculosis is a well-recognised phenomenon in adult life, but its exact significance has not been fully explained. In many cases, however, it appears to originate as an erosion consequent upon failure of the right side of the heart.

In adult life, gastric ulcer not infrequently occurs during the course of an acute infectious disease, and if we may judge from the cases which have been recorded, children are by no means exempt from a similar affection as the result of pneumonia, diphtheria,[1] variola,[2] and enteric fever. In the case of the last-named disease the gastric ulceration may sometimes arise from necrosis of the solitary glands, but in other instances it is probably a result of the catarrhal condition of the mucous membrane of the stomach which so often accompanies the onset of an infectious fever.

In one instance the gastric disease was associated with general septicæmia arising from a gangrenous affection of the mouth, and in a similar case recorded by Klebs[3] the mucous membrane of the stomach was found to present numerous small patches of gangrene after death. It is possible that in such cases the gastric disease was due to the presence of micrococci in the tissues of the organ.

The fact that ulceration of the duodenum occasionally follows burns of the skin has been frequently attested since the publication of Curling's original memoir, but it is not generally understood that the stomach may also exhibit a similar lesion. That gastric ulcer does occasionally ensue from superficial burns is shown by the case which has just been related (Case 11), as well as by the fact that the association of the two diseases is noted more than once in the records of the London Hospital. In every case of severe burn which I have examined, the microscope demonstrated the existence of gastro-intestinal catarrh, the signs of which were usually most pronounced in the upper part of the small intestine. This condition was often associated with swelling of the solitary lymphatic glands, and not infrequently also with punctiform hæmorrhages in the stomach and duodenum. In one case, where several superficial ulcers were found in the first part of the small bowel, the edges of the sores afforded unmistakable evidences of an hæmorrhagic origin. It is probable, therefore, that the catarrhal process may occasionally give rise to interstitial hæmorrhages, which in due course are converted into definite ulcers.

---

[1] Malinowski (*Index Medicus*, vol. v., p. 575) found an ulcer in the stomach of a child ten years old, who died of diphtheria, and Rilliet and Barthez (*op. cit.*, 2nd edit., vol. ii., p. 881) describe a somewhat similar case.

[2] Charcot (*Mém. de Soc. Biol.*, 1853, p. 88) found two ulcers in the stomach of a six-months fœtus which was affected with small-pox.

[3] *Virchow's Archiv.*, vol. xxxii., p. 198.

With regard to the etiology of this gastro-duodenal catarrh, it has been shown by Hunter[1] that the subcutaneous administration of toluylenediamine to dogs is apt to be followed by severe duodenitis with ulceration in the vicinity of the bile-ducts, and he has consequently propounded the theory that in cases of burns some irritant substance is eliminated by the bile, and produces a local effect upon the duodenum. There are, however, two facts which appear to me to militate strongly against this view. In the first place, it has already been stated that the inflammatory condition is not confined to the first part of the intestine, but also affects the stomach; so that if the theory of local irritation is accepted, it is necessary to believe that the bile habitually gains access to the stomach—a supposition which is not borne out by pathological experience. In the second place, while I have been able to confirm the statements of Hunter with regard to the effects of toluylenediamine, I have also found that the same results can sometimes be produced after preliminary ligature of the bile-duct; indeed, in one case two large perforating ulcers were found in the duodenum within thirty-six hours of the administration of the drug. In two cases of severe burns, I collected the various sloughs with some of the purulent secretion from the sores, and immersed them in absolute alcohol for several weeks, after which the coagulated proteid matter was extracted with distilled water. After several subsequent precipitations with absolute alcohol, a white powder was obtained which gave most of the reactions of an albumose; and this, when injected beneath the skin of two cats, gave rise to an appreciable degree of gastro-duodenitis. Unfortunately, the supply of the material was very small, and I was not able to repeat the experiment with larger doses; but I cannot help thinking that further investigation into the matter might be productive of interesting results.

---

[1] *Path. Soc. Trans.*, vol. xli., p. 105.

Considering the important excretory functions performed by the alimentary canal, it appears to me to be more probable that the mucous membrane of the duodenum and stomach is capable of eliminating toluylenediamine and other poisons from the system, but that in so doing it is liable to fall a victim to its own activity, and to become affected with acute inflammation.

Experimental pathology teaches that all forms of abrasion of the mucous membrane of the stomach tend to undergo spontaneous and rapid repair as soon as their exciting cause has been removed. Thus, in whatever manner a gastric ulcer is produced in an animal, the sore heals completely within a few days;

and it is only by the adoption of certain elaborate precautions that chronic ulceration can be established. This remarkable tendency to repair is not confined to the stomachs of inferior animals, for there are several facts which indicate that the same aptitude for healing also exists in the human subject. Thus, mechanical injuries to the gastric mucous membrane frequently occur in persons who earn their livelihood by swallowing swords, knives, and other sharp instruments, yet chronic ulceration from such a cause is almost unknown; while large pieces of the inner coat of the stomach are occasionally torn away by careless manipulation of the stomach-pump without the production of any subsequent ill effects. Again, severe hæmatemesis associated with the symptoms of gastric ulcer is by no means uncommon in girls between the ages of seventeen and twenty-five, and has been observed several times in young children.[1] Nevertheless, when the stomach is placed in a condition of comparative physiological rest, a rapid and complete cure usually ensues. Lastly, scars and other evidences of former ulceration are frequently encountered in the post-mortem room, and Donné[2] has recorded a case where he found a healed ulcer in the centre of the lesser curvature in a child three years of age. The main question, therefore, in the etiology of *chronic gastric ulcer* is why an abrasion of the mucous membrane fails to heal.

The causes which prevent the repair of an ulcer of the stomach may be divided into two classes, local and general, the former being, in most instances, of the greater importance.

---

[1] Wertheimber, *Jahrb. f. Kinderheilk.*, 1883, s. 79.

[2] Cited by Rilliet and Barthez, *op. cit.*, p. 883. I have also seen a typical scar in the stomach of a girl aged sixteen, who had previously suffered from 'dyspepsia.' It would appear that perforating ulcers always occur at an earlier age in girls than in boys.

Local Conditions which prevent Healing.

1. *Situation.*—A glance at the various cases which have been recorded is sufficient to show that, even among the manifold differences which exist between the acute and chronic forms of the disease, the anatomical situation of the ulcer is by no means the least remarkable. In almost all the cases of the acute variety the disease occupied the cardiac or middle zone of the stomach near the great curvature; but in four out of the five cases of the chronic complaint where the exact position of the ulcer is recorded, it was found to be situated in the pyloric end of the organ, near the lesser curvature—in fact, at the same spot where chronic ulcer usually occurs in adult life.

It is a well-known fact that persons who survive the immediate results of swallowing corrosive fluids, such as carbolic, nitric, and hydrochloric acids, often exhibit after death a diffuse scarring of the mucous membrane in the cardiac and middle zones of the stomach, with an open and chronic ulcer near the pyloric orifice. It can also be demonstrated by experiment upon gastrostomized animals, that mechanical injuries inflicted upon the mucous membrane in the region of the pylorus heal much less rapidly than those which affect the fundus of the organ.

Among the many causes which tend to prevent healing of a sore in the pyloric region, *want of rest* doubtedly ranks first. In surgical practice the diffi of healing an ulcer of the skin situated in the neighbourhood of a large joint can be successfully overcome by the application of splints. But in the case of the stomach it is impossible to prevent the constant opening and closure of the pyloric orifice, and so to afford the injured tissue a period of complete rest. Another factor which helps to prevent the repair of a pyloric ulcer is the firm adhesion which exists between the mucous membrane and the muscular coat in this region of the stomach. In the fundus and central portions of the organ the submucous connective tissue is loose and abundant; but in the neighbourhood of the pylorus it is extremely scanty, and firmly binds the inner coat to the subjacent tissues, with the object of preventing its prolapse through the aperture when the stomach contracts. This anatomical condition is therefore comparable to that which exists over the inner surfaces of the tibiæ, where the absence of subcutaneous tissue causes an ulcer of the skin to become adherent to the periosteum, and consequently to heal with difficulty.

2. *Deficient Blood-supply.*—As a rule, children are not affected by those arterial diseases which exert such an important influence upon the development of a gastric ulcer in later life. It is probable, however, that a deficient or irregular circulation of the blood in the region of the pylorus is partly responsible for the tardy repair of abrasions in this region of the stomach. When the human stomach is artificially injected, it at once becomes evident that the mucous membrane in the cardiac and middle zones of the viscus habitually receives the greater portion of blood which enters the organ; for in many cases I

have found that the tissues in the pyloric region were only partially injected when the rest of the stomach was fully engorged with the colouring fluid. This peculiar vascular distribution is in strict accordance with the physiological law which ordains that the blood-supply of a tissue should be strictly proportionate to its functional activity, since it is the mucous membrane of the fundus and central portion of the stomach which is chiefly concerned in the secretion of gastric juice and the absorption of the products of digestion. It is also to be observed that the vessels supplying the pyloric region of the stomach pierce the muscular tissue in an oblique manner, and are consequently subjected to compression at each contraction of the wall of the viscus. This latter fact may be easily demonstrated by passing a strong electric current through the stomach, when the mucous membrane in the vicinity of the pylorus is observed to become quite anæmic.

3. *Hyperacidity.*—Of late years considerable stress has been laid upon the abnormally acid condition of the gastric juice in cases of gastric ulcer, and efforts have been made to trace a causal connection between the hyperacidity and the organic lesion. Although hyperacidity often coexists with chronic ulceration of the stomach, the association of the two complaints is not invariable, nor does the organic lesion occur in more than a small proportion of the cases of simple hyperacidity. It has, however, been shown by experiment that an excessive acidity of the contents of the stomach tends to stimulate the process of ulceration, and causes the disease to assume a chronic character. Consequently, hyperacidity must be re-

garded as an important factor in the prevention of repair of an ulcer of the stomach.

4. *Adhesions.*—As soon as an ulcer has contracted adhesions with some solid organ, such as the liver or pancreas, the chance of complete cicatrization becomes comparatively remote, since the edges and base of the sore are unable to contract. Even in these cases, however, an attempt at repair may often be observed in those parts of the ulcer which are the least fixed.

Constitutional Causes of Non-Healing.

The clinical fact that anæmia frequently accompanies the process of gastric ulceration appears to indicate that some morbid condition of the blood may possibly prevent repair of the mucous membrane of the stomach. This conclusion has received considerable support from the experiments of Daettwyler,[1] Silbermann,[2] and others, who have shown that if an animal is rendered anæmic previous to the production of an ulcer in its stomach, the injury inflicted upon the mucous membrane, instead of healing, will often assume a chronic and progressive character. It is also probable that the cachexia which accompanies such diseases as phthisis, syphilis, and malaria is occasionally responsible for the non-healing of a simple gastric ulcer, for it is a common observation that in such cases the gastric disease will refuse to heal until after the administration of iron, arsenic, or iodide of potassium.

---

[1] *Correspondenzbl. f. Schweizer Aerzte*, 1875, s. 101.

[2] *Deut. Med. Woch.*, 1886, 29, s. 497.

4. TUBERCULAR ULCER.

Tubercular affections of the stomach are somewhat less rare in childhood than in adult life. In cases of general miliary tuberculosis, a few gray or yellow tubercles are often found scattered through the submucous tissue in the pyloric region of the organ, but these seldom proceed to ulceration; more commonly caseation takes place without destruction of the mucous surface.

True tubercular ulceration of the stomach is usually associated with a similar disease of the intestine, or with chronic phthisis. Steiner and Neureutter[1] met with four instances of the disease among 302 autopsies on tuberculous children, and Widerhofer[2] noted it twice in 418 cases. The observations of Rilliet and Barthez[3] would appear to denote that this variety of ulceration of the stomach is more common than is usually believed, since they found it in no less than twenty-one out of 141 cases of tubercular disease of the gastro-intestinal tract. It must be noticed, however, that these latter authorities seem to have taken the tuberculous nature of the disease for granted in many cases, for they distinctly state that in fourteen instances out of the twenty-one no tubercle was observed. It is quite probable, therefore, that many of their cases were examples of the simple, rather than the tuberculous, variety of gastric ulceration.

As a rule, the ulcer varies from two or three lines to several centimetres in diameter. Its favourite seat is in the pyloric end of the organ, near the lesser curvature, and it is often multiple. In shape it varies considerably; in some cases it is circular, in others oval; while in not a few the outline is irregular and presents a scalloped appearance. The edges are usually raised above the surrounding tissue, and are thickened and nodular; but occasionally the disease may closely simulate in its general features the simple perforating ulcer. The base is shallow, and generally formed by the submucous coat, and presents a yellowish tinge and granular aspect. In many cases a number of discrete tubercles can be observed scattered through the mucous membrane in the immediate vicinity of the sore. The peritoneal aspect of the disease almost always exhibits a few miliary tubercles, and not infrequently the base of the ulcer is adherent to some neighbouring organ, such as the liver, pancreas, or colon. The lymphatic glands situated in the retroperitoneal tissue and along the lesser curvature are enlarged and caseous.

---

[1] Cited by Rehn, *op. cit.*

[2] Gerhardt's 'Handbuch,' vol. iv., s. 444.

[3] *Op. cit.*, vol. iii., p. 1262

On microscopical examination, the edges and base of the ulcer present the typical tubercular structure, in which both giant cells and bacilli are easily demonstrated.

This variety of ulceration seldom gives rise to hæmorrhage or terminates in perforation, owing to the gradual obliteration of the vessels and the numerous adhesions which form round the base of the disease. Indeed, in the literature upon the subject, I have only been able to find one instance where perforation is stated to have occurred, and in this case the tubercular origin of the gastric lesion appears to be somewhat doubtful.[1] Occasionally an encysted collection of pus arising from tubercular peritonitis discharges itself through the walls of the stomach;[1] and there are a few cases on record where a caseous lymphatic gland in the neighbourhood underwent suppuration, and finally burst into the cavity of the viscus.[2]

---

[1] Paulicki, *Berl. Klin. Woch.*, 1867, s. 349.

The comparative immunity from tubercle which the stomach enjoys appears to depend upon two principal causes. In the first place, unlike the intestine, the stomach only contains a small amount of lymphoid tissue, which is deeply situated in the substance of the mucous membrane. In the second, the acid character of the gastric juice, though it may not actually destroy the bacilli, is distinctly inimical to their growth, and hence, even when they are introduced in large quantities into the stomach by means of the swallowed expectoration, they are rendered temporarily inert, and are passed into the intestine without having effected a permanent lodgment.

5. *MALIGNANT DISEASE.*

There appear to be only two authenticated cases of carcinoma of the stomach in children less than fourteen years of age. The first was reported by Cullingworth,[3] who found a pear-shaped tumour, about an inch in length, growing from the pyloric extremity of the stomach in an infant five weeks old. Microscopical examination of the growth proved it to be a cylindrical cell epithelioma. In the second case, which is recorded by Scheffer,[4] an encephaloid tumour was found in the stomach of a boy fourteen years old, which had given rise to secondary deposits in the spleen and mesenteric glands.

---

[1] Bignon, 'Thèse de Paris,' 1884.

[2] Besnier, *Soc. Anat.*, 1857; Pitt, *Path. Soc. Trans.*, 1888, p. 107.

[3] *Brit. Med. Jour.*, 1877, vol. ii., p. 253.

[4] *Jahrb. f. Kinderheilk.*, 1880, s. 425.

Kaulich[1] has also described colloid cancer of the abdominal viscera in a child a year and a half old, but in this case it is extremely doubtful whether the disease commenced in the stomach. The case recorded by Williamson,[2] which is usually cited as gastric carcinoma, was most probably one of congenital hypertrophy of the pylorus (p. 315). Jackson[3] has related a case of cancer of the stomach in a boy fifteen years of age.

SYMPTOMS OF GASTRIC ULCER.

The symptoms of gastric ulceration in childhood vary according to the age of the patient and the character of the disease. In young infants who are unable to express their subjective sensations, the first thing to attract attention is usually the occurrence of hæmatemesis or melæna. In older children, dyspepsia or abdominal pain usually precedes the development of the more characteristic phenomena of the complaint, but even in these cases the gastric symptoms are often masked by others arising from the primary disease (tuberculosis, typhoid, septicæmia, etc.).

Dyspepsia.—It is interesting to observe that, in three out of the five cases of chronic ulcer, the symptoms of the disease were so slight and variable in their nature as to merit the description of 'dyspepsia' or 'indigestion.' In these cases discomfort at the epigastrium after meals, with nausea, flatulence, and constipation, appear to have been the sole symptoms of the complaint, until an accidental catastrophe, like hæmatemesis or perforation, demonstrated the real nature of the disease. There are several other cases on record where severe hæmatemesis occurred during the course of so-called dyspepsia, so that it would appear that 'latent' gastric ulcer is by no means a phenomenon which is totally confined to adult life.

---

[1] *Prag. Med. Wochen.*, 1864, No. 34.

[2] *London and Edinburgh Monthly Journal*, 1841, p. 23.

[3] *Proc. Boston Soc. for Med. Improvement*, 1867, App., p. 109.

Pain.—Pain in the epigastrium after food is stated to have occurred in three cases, and in one instance (Case 18) it is described as having been extremely severe. It usually commences soon after a meal, and persists during the whole period of digestion or until emesis takes place. Exacerbations are apt to be experienced from time to time, and usually indicate an extension of the ulceration. A violent attack of pain not infrequently precedes hæmatemesis or perforation. As a rule, the morbid sensation is referred to one spot in the epigastrium, but occasionally it is located in the spine, near the last dorsal or first lumbar vertebra. In almost every instance there exists a tender spot just below the tip of the ensiform cartilage, and pressure over this area is apt to excite nausea and vomiting. General tenderness of the epigastrium is sometimes encountered in cases of diffuse follicular ulceration or of hæmorrhagic erosions associated with acute gastric catarrh.

Vomiting.—This is a variable symptom of gastric ulcer, and was only observed in three cases. It seldom occurs immediately after food, but generally takes place at the culminating point of a painful crisis. It is preceded by nausea, and not infrequently also by a profuse secretion of saliva. The emesis has the eff of relieving the abdominal pain by ridding the stom of its acid contents. In cases of follicular ulceration, all forms of food are rejected almost as soon as they are swallowed.

In most cases the ejecta are acid in reaction, but it has not yet been shown that they contain an abnormal amount of free hydrochloric acid. Occasionally minute pieces of mucous membrane may be recognised in the vomit in cases of hæmorrhagic erosion of the stomach.

Hæmatemesis.—Although vomiting of blood is always suggestive of ulcer of the stomach, it must not be regarded as pathognomonic of that disease. Infants at the breast occasionally vomit small quantities of blood mixed with curdled milk, but in most instances the source of the bleeding is to be found either in a cracked state of the nipples, or in an ulcerated condition of the infant's gums and mouth. Spurious hæmatemesis is also apt to arise from post-nasal hæmorrhage, while sometimes hæmoptysis is confused with bleeding from the stomach, owing to the fact that children habitually swallow their expectoration. Even in the absence of these obvious sources of hæmorrhage, the mere presence of blood in the vomit must not be regarded as necessarily indicative of ulceration of the stomach. Thus, the severe retching which follows the paroxysms of whooping-cough is often accompanied by the rejection of a small quantity of bright blood, derived from the vessels of the pharynx or tongue, while in such diseases as purpura, scurvy, hæmophilia, and jaundice, which are characterized by hæmorrhages from various mucous membranes, blood may be effused from any part of the digestive tract. Lastly, it may be mentioned that in many of the malignant specific fevers, notably in measles and variola, genuine hæmatemesis is occasionally observed as a precursory symptom of death.

Hæmatemesis was noted in four cases of the acute and two cases of the chronic form of gastric ulcer in children. As a rule, the symptom shows itself shortly after a meal, but sometimes active exercise or a sudden emotion appears to determine an attack. The quantity of blood which is vomited bears no relation to the size of the ulcer, since it is the depth rather than the superficial extent of the disease which endangers the integrity of the gastric vessels. When the blood is considerable in amount, it usually possesses a bright-red colour, and indicates that a large vessel has been eroded; but when the vascular leak is small, the vomited matter is black or brown in colour, owing to the action of the gastric juice upon the hæmoglobin. It must always be borne in mind that a child may continue to bleed internally after the hæmatemesis has apparently subsided, the nervous depression induced by the hæmorrhage preventing a recurrence of the emesis. Under these conditions the patient grows anæmic and restless, the pulse increases in frequency while it loses in strength, and death ensues from collapse or syncope. In four of the recorded cases the stomach was found to be full of blood after death.

Melæna.—Hæmorrhage from the bowel is apt to occur in all cases of true hæmatemesis, and in some instances may even completely replace the vomiting of blood. It may also arise from most of the conditions which have already been noted as productive of spurious hæmatemesis, and is frequently encountered in chronic intestinal catarrh, dysentery, typhoid fev intussusception, cirrhosis of the liver, piles, polypi. There is, however, one special variety which, from its occurrence soon after birth, is known as 'melæna neonatorum.' The pathology of this affection is rather obscure, and many theories have been advanced to explain its origin. Thus, Billard[1] believed that it was due to the follicular ulceration of the stomach which he had so often observed in young infants, while Landau[2] and others have ascribed it to embolism of the umbilical vein, or to fatty degeneration of the bloodvessels. When the disease proves fatal soon after birth, the bloodvessels of the abdominal viscera are found at the necropsy to be greatly engorged, and in not a few cases hæmorrhagic erosions of the stomach or duodenum have been observed. In other cases the source of the bleeding has not been discovered, and in such hæmophilia is often regarded as responsible for the effusion. In one case of fatal hæmorrhage which came under my notice, post-mortem examination failed to explain the origin of the bleeding, but when the vessels of the stomach were subsequently injected, a large vein was discovered close to the cardiac orifice, through an aperture in which the injection poured out in great quantities. It is possible, therefore, that in many cases where the cause of the bleeding is not detected, post-mortem contraction of the mucous membrane may have obliterated the signs of rupture of some varicose vein in the stomach or bowel. This supposition is supported by the fact that in a great many cases the hæmorrhage ensues after a tedious labour, or where the respiratory functions have been established with great difficulty—conditions which eminently dispose to severe congestion of the gastro-intestinal tract and rupture of a dilated vessel.

---

[1] *Op. cit.*

[2] 'Ueber Melæna,' Habilitationsschrift, Breslau, 1875.

The bleeding usually commences within forty-eight hours of birth, but occasionally it may not appear for several days. Thus, among the twenty cases recorded by Rilliet and Barthez,[1] nine exhibited melæna within thirty-six hours, and seventeen before the sixth day; while in twenty-seven out of the thirty-seven cases collected by Silbermann,[2] the hæmorrhage occurred within the first two days.

The first sign of severe intestinal hæmorrhage is usually anæmia with collapse. This is followed by the evacuation of a motion consisting of thick. dark blood, after which the infant becomes breathless and very restless. The stools succeed one another in rapid succession, and soon consist entirely of bright blood. The hæmorrhage often continues for twenty-four hours, and is not infrequently accompanied by hæmatemesis. If the bleeding is profuse, the collapse soon becomes profound, and the infant succumbs to asthenia, syncope, or to respiratory failure after a series of convulsions. In less severe cases it gradually improves, but it may remain anæmic and feeble for many weeks, or eventually die of acute intestinal catarrh. The mortality from melæna neonatorum is about sixty per cent.

Other Symptoms.—The *tongue* is usually clean, red, and pointed. If, however, gastric catarrh complicates the ulceration, the organ is thickly coated and the breath offensive. Flatulence is not a common symptom, but *acidity* and *pyrosis* are present. The *appetite* is seldom adverse and the patient's abstention from food is more often due to dread of the pain than to actual want of relish. The *bowels* are confined, and the stools hard and knotty, and not infrequently enveloped in a thick coating of mucus.

---

[1] *Op. cit.*, 2nd edit., vol. ii., pp. 295-310.

[2] Gerhardt's 'Handbuch,' iv., s. 41

Like all other chronic diseases of the stomach in early life, the presence of ulceration exercises an important influence upon the processes of growth and nutrition. In most instances the osseous and muscular tissues cease to grow, and the child becomes emaciated and stunted in stature. These latter phenomena are well illustrated by Case 18, where the patient at the age of eighteen is said to have been so extremely thin and diminutive in appearance as to resemble a boy of ten.

COMPLICATIONS.

Perforation of the stomach occurred in four out of the nineteen cases which have been recorded at length. The first symptoms of this accident consist of severe pain at the epigastrium, and vomiting, followed in a short time by collapse. In one instance (Case 9) the extravasation of the gastric contents into the cavity of the peritoneum gave rise to convulsions. If life is prolonged for a few hours, the signs of acute general peritonitis develop. In this condition the child lies upon its back with the knees drawn up, and is afraid to move, or even to speak, on account of the abdominal pain. The face is pale and covered with sweat, the pulse quick, small, and wiry, and the breathing rapid and costal in character. The abdomen is tense from rigidity of the superficial muscles, and the slightest pressure gives rise to severe pain. The bowels are obstinately confined, and from time to time a small quantity of green bile is pumped up into the mouth without effort. Death usually occurs within thirty-six hours. Perforation of the diaphragm, with subsequent pyo-pneumothorax, such as occurred in Case 19, is at present a unique result of gastric ulceration in childhood. Dilatation of the stomach can only occur when a chronic ulcer is situated close to the pylorus, as in Case 18. When this complication arises, the previous history of the case, in conjunction with the physical signs of gastric dilatation and the characteristic vomit, are sufficient indications for an accurate diagnosis. Up to the present time neither hypersecretion nor tetany have been observed in cases of ulceration of the stomach in early life.

It may not be out of place here to say a few words upon the subject of *congenital stenosis of the pylorus.* The first case on record was probably that which was erroneously described by Williamson[1] as 'scirrhus.' and has since been cited as an instance of cancer of the stomach in infancy. Landerer,[2] in 1879, gave a good account of the disease, and at a later date Maier[3] collected thirty-one cases. Since that time many other instances of the complaint have been recorded,[4] and two cases have come under my own observation. After death the pylorus is found to be considerably thickened owing to hypertrophy of its circular or longitudinal muscle fibres, and its orifice stenosed. In long-standing cases the stomach is usually dilated, and its mucous membrane shows signs of chronic catarrh. The disease usually proves fatal within the first three months of life, and often within a few weeks.[1] In some of the recorded cases, however, life appears to have been prolonged for nearly two years. As a rule, vomiting does not occur for several days after birth, but when it has once set in, it takes place after every meal, and the infant commences to lose flesh and strength. Constipation is a marked feature, and the stools are extremely scanty. In one instance I was able to examine the contents of the stomach after a test-meal, and found that the secretion of hydrochloric acid was normal. When dilatation of the stomach, accompanied by catarrh of the mucous membrane, ensues, the acid secretion diminishes. Finkelstein[2] has observed a case where the thickened pylorus could be felt during life as a small cylindrical tumour above the umbilicus, and in the second case which came under my care the same physical sign was present. In most instances medical treatment is useless, but in chronic cases associated with dilatation of the stomach, it is possible that resection of the pylorus might be undertaken with success.

---

[1] *London and Edinburgh Monthly Journal*, 1841, p. 23.

[2] 'Ueber Angeborene Stenose des Pylorus,' Dissert., Tübingen, 1879.

[3] *Virchow's Archivs*, 102, s. 413.

[4] Pitt, *Path. Soc. Trans.*, 1892, p. 63; Hirschsprung, *Jahrb. f. Kinderheilk.*; Peden, *Glasgow Med. Journ.*, June, 1889, p. 416.

TREATMENT.

General.—Rest in bed is essential in all recent and acute cases. In the chronic form, or in those where the more urgent symptoms have subsided, a moderate amount of daily exercise may be permitted. Whenever there is much tenderness on pressure, the application of hot fomentations or poultices to the epigastrium is found to afford relief; but if the child is strong and otherwise healthy, two or three leeches applied to the affected part are usually of greater service. In chronic cases, considerable benefit is usually derived from the repeated use of small blisters over the epigastrium.

---

[1] Thompson, *Edinburgh Hospital Reports*, vol. iv., 1896.

[2] *Jahrb. f. Kinderheilk.*, Bd. xliii., i., s. 105.

Dietetic.—If the infant is at the breast and the milk agrees, it should be nursed every hour, but only permitted to imbibe a small amount of milk on each occasion. In the case of hand-fed children, cow's milk sterilized and diluted with barley or Vichy water may be allowed with the same precautions. Should this disagree, the milk may be peptonized or humanized before its administration (Chapter II.). Occasionally every form of milk is rejected, unless previously mixed with malted flour or some other farinaceous material. In older children, milk should still form the staple diet, but it may be alternated occasionally with strong soups, extract of meat, raw meat juice, or meat jellies. As soon as improvement has set in, lightly-boiled eggs, white fish, boiled chicken, sweetbread, or finely-scraped and lightly-cooked meat, may be cautiously tried.

Medicinal.—Opium is of the greatest value in all cases, since it relieves pain, controls peristaltic action, and diminishes the gastric secretion. The tincture is usually the best preparation for infants, and may be administered in half-minim doses twice or thrice a day. In other cases, chlorodyne or nepenthe answers equally well; while in older children the solid extract of opium in doses of a quarter of a grain is to be preferred. If gastric intolerance is a prominent symptom, a mixture containing carbonate of bismuth, carbonate of sodium, and two minims of the solution of morphine, given before meals, is of great value.

In chronic cases, the preparations of copper and silver are occasionally of value, but serious results are apt to follow their premature administration. As soon as improvement has set in, recourse should be had to arsenic, iron, or cod-liver-oil.

In cases of 'melæna neonatorum,' the infant must be kept absolutely at rest, and all forms of nourishment artificially cooled. In most cases a minute dose of morphine is followed by the best results, but in severe cases it may be necessary to administer a grain of gallic acid, combined with tincture of opium, in the form of an enema, or to inject half a grain of ergotin beneath the skin. Occasionally a minim of oil of turpentine suspended in mucilage, and given every hour, appears to exert a controlling effect upon the hæmorrhage. Stimulants must be avoided as far as possible, as they tend to excite the action of the heart and to increase the bleeding.

CHAPTER X.

THE DYSPEPSIA OF STRUMOUS CHILDREN.

IN the year 1833 Dr. T. J. Todd[1] described a variety of dyspepsia which he considered was peculiar to strumous children. A few years after the publication of this important memoir, Sir James Clark[2] again drew the attention of the profession to the subject, and not only fully confirmed the original description of the disorder, but even went so far as to assert that the gastric derangement was the most constant and typical feature of the scrofulous habit. Important as these observations were, it is open to doubt, in the light of our present-day knowledge, whether some of the cases described under this title were not in reality examples of rickets, and the digestive disorder which was attributed to the influence of scrofula immediately dependent upon intestinal catarrh.

Of late years the association of dyspepsia with the tubercular diathesis has attracted but little notice, and modern writers are content either to quote the opinions of Clark, or to dismiss the subject with the brief remark that dyspepsia is often encountered in strumous children.

---

[1] 'Cyclopædia of Practical Medicine,' 1833; art. 'Indigestion,' vol. ii., p. 649.

[2] 'Pulmonary Consumption,' 1835, p. 16.

My attention was first attracted to this disorder three or four years ago by the case of a young lady who was brought to me on account of violent attacks of abdominal pain, associated with symptoms of deranged digestion, and since that time I have seen many instances of a similar nature. The complaint is encountered in a modified form in almost every child who presents the general features of the tubercular diathesis, but it is comparatively rare for the disorder to prove so severe as to claim the exclusive attention of the practitioner. I find that, among six thousand cases of disease in children which have come under my care during the past four years at the Evelina Hospital, fifty-seven were brought to me solely on account of the complaint in question, and it is principally from observations made upon these cases that the following remarks have been compiled.

SEX AND AGE.

Girls appear to be much more subject to the disease than boys, for among my fifty-seven cases no fewer than forty-four were of the female sex. The disorder may exhibit itself at any time between the third and fourteenth years, but it is most common about the age of five or six. In only five of my cases had the child reached its twelfth year before the complaint came on, but I have seen two or three cases in which the first symptoms appeared after puberty.

FAMILY HISTORY.

In the vast majority of the cases which come under observation, the patient exhibits a strong family tendency to tuberculosis. Thus, in about 66 per cent.

of the cases it was found that one or other of the parents had suffered from phthisis, and it is interesting to notice that in the majority of these it was the father who was the subject of the pulmonary disease. In nearly one-third of the entire number, it was stated that one or other of the grandparents had also suffered from the same complaint.

Family predisposition to acute rheumatism was pronounced in seven instances, while in three others 'rheumatic gout' was said to exist in some near relative. In one case a sister had suffered from chorea, but with this exception the patients who have come under my observation were free from any neurotic taint. Todd believed that the offspring of old men were particularly liable to fall victims to this form of dyspepsia, but this statement I have not been able to confirm.

As a rule, one or more of the patient's family exhibit evidences of the strumous diathesis. In eighteen cases I found that one of the other children was either the subject of enlarged cervical glands, or possessed scars or other signs of disease of the lymphatic system or the bones.

GENERAL APPEARANCE.

The digestive disorder affects all varieties of the so-called scrofulous temperament, but the children who possess the finer and more delicate caste of features prove the most frequent sufferers.

In addition to the general appearance indicative of the diathesis, some local manifestations of scrofula are occasionally encountered. Chronic enlargement of the cervical or submaxillary glands was only noted in three cases, but in four others typical scars were observed. Chronic inflammation, with hypertrophy of the tonsils, existed in seventeen, and phlyctenular ulcers of the cornea in eight cases; but tubercular disease of the long bones or joints was only once observed. Otorrhœa was frequently present, and many of the children suffered from muco-purulent discharges from the nose or vagina without any obvious disease being detected in these organs. Anæmia was an invariable symptom; and the digestive derangement seldom underwent any decided improvement until the conjunctivæ and the mucous membranes began to regain their normal colour.

SYMPTOMS AND PROGRESS.

Pain in the abdomen constitutes one of the most constant and characteristic features of the disease. It is usually quite sudden in its onset, and is apt to recur at irregular intervals. When the child is seized with the pain during the course of the day, it may be noticed suddenly to cease its play and to clasp its hands over the abdomen, while not infrequently it will throw itself upon the ground and appear to writhe in agony. Should the attack occur during sleep, the patient awakes with a scream, and jumps up in bed with an expression of the greatest possible alarm. Sometimes the face becomes blanched before the onset of the pain, and the sudden pallor is sufficent to warn the nurse of the approach of an attack; in other cases the face is deeply flushed, the conjunctivæ injected, and beads of perspiration appear on the forehead and neck.

As a rule, the attack commences during the early part of the evening or night (seven to nine o'clock), but occasionally it is postponed until six or seven in the morning. The majority of the cases also suffer from a recurrence of the symptom during the course of the day, especially about noon. In about 52 per cent. of my cases the ingestion of food was followed within a few minutes by severe abdominal pain, and the patient was often obliged to hurry off to the closet, owing to a sudden and urgent call to empty the bowels. The umbilical region is the part of the abdomen to which the pain is usually referred, though occasionally other districts are also affected. The symptom partakes of the nature of a cutting or twisting sensation, and it frequently appears to commence a little to the right and slightly above the navel, and to travel transversely across the abdomen toward the left. Occasionally, however, the patient has assured me that the pain proceeded in the reverse direction, or remained situated in the hepatic or splenic region. In a few cases the hypogastrium was indicated as the chief seat of the suffering.

The pain may prove severe from the very commencement of the attack, but it generally grows more and more intense until a maximum is reached, after which it gradually declines. Sometimes the expulsion of a large quantity of gas either by the mouth or the rectum is followed by more immediate relief.

The actual duration of the pain varies from five minutes to several hours. The nocturnal attacks are the most severe, and endure the longest; but when the disease is abating their duration is much curtailed, and they may eventually be replaced by a mere momentary twinge.

The affected region of the abdomen is sometimes seizure, and in one obstinate case, where the attacks occurred on the same night each week, it was found on inquiry that the child had to accomplish a considerable amount of arithmetic before going to bed on that evening, a task which always proved extremely wearisome. As soon as this error was remedied, rapid improvement took place, and the pain eventually disappeared.

The condition of the *appetite* varies considerably. Todd and Clark found that the desire for food was often insatiable, and this I have also occasionally observed. As a rule, however, the appetite is poor and exceedingly capricious, and the child is apt to develop peculiar distastes for certain articles of food. Meat fat is particularly repugnant to many of these patients, and I found that no fewer than 87 per cent. of my cases were unable to eat this substance without experiencing nausea, acidity, vomiting, or an attack of 'biliousness.' This dislike is sometimes so intense that the mere sight of fat gives rise to a feeling of nausea. Bacon fat, on the other hand, is often eaten with pleasure, and milk, cod-liver oil, and glycerine are easily tolerated.

As a rule, children are extremely partial to sweet things, but 30 per cent. of my cases of this disorder disliked sugar in any form, and the mother usually stated that saccharine substances made them 'bilious.'

In a few instances the statement was volunteered that the child disliked the ordinary kinds of food, bu was very fond of indigestible materials. Inquiry in this subject generally elicited the fact that all sour a acid substances were regarded as special
Thus, in one instance the mother complai

daughter had ruined her digestion by inordinate indulgence in vinegar, while in another the child was said to almost subsist upon lemons. These facts are interesting, since the same tastes are often exhibited by patients suffering from the dyspepsia of early phthisis.

Thirst usually constitutes a prominent symptom of the disorder, and was present in 63 per cent. of my cases. The sensation is chiefly complained of at night, and the child will often get out of bed and search for water to drink; and in one instance I was informed that the dirty contents of the hand-basin had been imbibed, in the absence of drinking-water from the room. Occasionally the thirst persists all day, and in these cases the appetite is very defective.

Constipation was present in nearly 70 per cent. of the entire number. In some cases a torpid condition of the bowels had existed throughout life, but as a rule the symptom had only shown itself for the first time shortly before the onset of the pain. The stools are pale and fœtid, and usually consist of a putty or mortar-like substance; but occasionally they are hard and knotty, or they may present the appearance of slime. On careful examination, the evacuated material is found to consist largely of unaltered food containing very little bile pigment—appearances which seem to justify the frequent observation of the nurse, to the effect that the food 'goes through the body unchanged.' Not infrequently a fœtid discharge takes place from the rectum, and occasionally small quantities of pure blood may be voided. I have never been able to discover hæmorrhoids or any other disease of the rectum to account for these symptoms, which usually disappear spontaneously as the case improves.

In about one-third of the cases the bowels were stated to be irregular in their action. In many of these it was a constant complaint that, as soon as a few mouthfuls of food had been swallowed, the child was attacked with pain in the abdomen, and had to hurry away in order to pass a motion. This sequence of events ensued after every meal, and was a cause of great annoyance both to the patient herself and to her parents. The evacuations in these cases are usually semi-solid in character, but sometimes they are liquid, and frequently give rise to a sensation of heat or scalding at the anus, or are accompanied by tenesmus. Occasionally the abdominal pain subsides without an evacuation of the bowels. Hot liquids or spiced foods appear to excite the peristaltic action of the intestines most readily, but sometimes the ingestion of fat meat is held by a patient to be responsible for the sudden diarrhœa. Under ordinary circumstances *nausea* and *vomiting* are seldom complained of, while *flatulence* and *acidity* are equally rare.

Although there may be no actual loss of flesh, the child remains thin and anæmic, and never seems to gain ground. The skin feels harsh and dry, but profuse perspirations are apt to occur during the night. The hands and feet are habitually cold and blue, and are very liable to chilblains. Sleep is much disturbed by dreams, and the child often talks wildly, gesticulates, or grinds the teeth while it dozes. The urine is usually pale in colour, and may deposit a considerable quantity of phosphates. The tongue is generally clean, and of a darker red than normal, but occasionally it appears to be veiled by a thin coating of white fur, through which the papillæ can be discerned in the form of vivid red spots.

In some children these symptoms are apt to be replaced by others, arising from an attack of acute gastric catarrh. When this disorder supervenes, the child awakes in the morning with frontal headache and a foul taste in the mouth, and complains of extreme nausea. The appetite is in complete abeyance, but thirst is often excessive. The face appears pale and puffy, and dark areolæ surround the eyes. The breath is sour, and the dorsum of the tongue covered with a thick yellow fur, while the tip and edges are bright red in colour. Occasionally small patches of aphthous ulceration appear on the gums and palate, and give rise to considerable discomfort. The pulse may be slightly quickened, and the temperature of the body raised a point or two above the normal, but the degree of pyrexia is usually insignificant. Nausea is a constant symptom, and retching or vomiting follows every attempt to partake of food. Acidity and flatulence are rarely encountered. As a rule, a mild form of diarrhœa accompanies the gastric disorder, but in some instances constipation occurs. The urine is passed in diminished amount, and deposits a quantity of amorphous urates. These catarrhal attacks are apt to recur every few weeks, and generally last from two to five days. Sometimes I have noticed a sudden change in the atmospheric conditions to be followed by an attack, especially when damp and mild weather has set in after a spell of dry north-east winds. In a few cases certain articles of diet appeared to be responsible for the attack; and I have several times heard it stated that a small quantity of fat which had been eaten was the cause of the 'biliousness.'

After the age of puberty the various symptoms of the complaint usually subside, but the patient may still be subject to occasional attacks of gastric catarrh. In some instances, however, the disease slowly undergoes a form of evolution, and the stomach, rather than the intestine, becomes eventually the chief seat of the disorder (Chapter VI.). In these cases it is sometimes possible to trace the gradual development of a peculiar variety of dyspepsia which in young adults not infrequently precedes the development of pulmonary tuberculosis.

ETIOLOGY.

In the absence of post-mortem evidence, it is only possible to offer a general surmise concerning the origin of the various symptoms of the complaint.

Todd considered that the dyspepsia was caused by a deficient secretion of the bile, whereby the processes of digestion and assimilation of the food in the duodenum were permanently hindered. Although there can be little doubt that this explanation is partially correct, since the general appearance of the patient and the pale and fœtid condition of the stools attest a deficiency of the biliary secretion, there are reasons for believing that the liver is not the sole organ which is at fault. The peculiar situation and character of the abdominal pain suggest that this symptom arises from an irregular and spasmodic contraction of the colon—a supposition which receives some amount of confirmation from the fact that in many cases the pain is immediately followed and relieved by an evacuation of the bowels. But unlike ordinary cases of colic, the symptom does not appear to depend upon the ingestion of irritating materials, nor does it arise entirely from an overloaded state of the large intestine. On the contrary, it is most readily excited by such conditions as mental or physical exhaustion, which exert their action through the medium of the central nervous system, or by a process of reflex irritation, as when food is introduced into the empty stomach. The sudden and violent peristalsis which is brought about in these various ways tends to curtail the period of gastric as well as intestinal digestion, and to hurry the food through the bowel before it has had time to undergo the necessary chemical changes.

It is well known that the tubercular diathesis is closely associated with various forms of neurosis, and it is therefore not unreasonable to suppose that the subjects of this disorder may occasionally exhibit an extreme irritability of the nervous mechanism of the digestive tract, which will show itself in the form of sudden and painful peristaltic waves. I am consequently inclined to regard the dyspepsia which is prone to attack strumous children in the light of a motor and sensory neurosis of the gastro-intestinal tract, rather than as a functional disorder of the stomach.

TREATMENT.

General.—The predisposition to tuberculosis exhibited by these cases requires the adoption of special precautions with the view of maintaining the general health. The child must always be warmly clad, and exposure to cold and damp carefully avoided. As a rule, flannel or woollen clothing should be habitually worn next the skin, and at night-time a flannel suit may be advantageously substituted for the cotton garment. Tepid baths of salt-water are useful in maintaining a healthy action of the skin, and regular, but not excessive, exercise in the open air should be encouraged.

Sea-air is particularly beneficial in the treatment of these cases, and a few months' residence at Margate, Walmer, Bexhill, or some other health resort on the east or south-east coast is often followed by marked improvement. The more relaxing atmosphere of the South of England, on the other hand, is seldom suitable for the subjects of this disorder, owing to the tendency to gastric catarrh which the climate appears to induce.

Dietetic.—The selection of an appropriate dietary seldom presents much difficulty. All articles of food which contain a large percentage of indigestible material must be avoided, and hence vegetables should be given sparingly or withheld altogether. White fish, chicken, and tender meat are to be preferred to the coarser and richer varieties; and all condiments and highly-spiced foods should be forbidden on account of the excessive peristalsis which their ingestion is apt to engender.

Milk and cocoa may be given freely, but coffee and strong tea must be prohibited. Alcohol is seldom required, and the constant resort to brandy as a means of relieving the pain is strongly to be deprecated. When improvement has set in, a little bitter ale or stout with the meals is often of service, but these beverages seldom agree in the early stages of the complaint.

The meals should be taken at regular hours, and it is often wise to administer some bread-and-milk, tapioca, gruel, or other light food about half an hour before the child retires to bed.

Medicinal. — The most prominent indication for medicinal treatment consists in the regulation of the bowels. Todd recommended the administration of small doses of gray powder for this purpose, followed by a course of mild hepatic stimulants, such as rhubarb, taraxacum, and nitrate of potassium. From my own experience, I am inclined to regard these remedies as being chiefly of value when the tongue is thickly coated and nausea is a prominent symptom. As a rule, I have found that the extract of cascara sagrada is by far the most reliable drug in the treatment of the constipation, and usually prescribe ten to fifteen minims of the liquid extract, along with a drachm of maltine, night and morning. Occasionally small doses of aloine with nux vomica answer well, or some simple laxative, such as the confection of senna and sulphur, may be employed with advantage.

Drastic and saline purgatives must be avoided, since the exhaustion which is apt to ensue from their employment increases the tendency to attacks of pain. When the bowels are moved after each meal, sedatives should be administered in order to relieve the abnormal irritability of the intestine, and with this object small doses of nepenthe, compound tincture of camphor, or a solution of morphine, may be given, in combination with carbonate of bismuth or aromatic sulphuric acid. In many instances of this nature I have found that a mixture containing a few minims of nux vomica and dilute solution of nitric acid effect a rapid cure.

As soon as the bowels have been brought into a satisfactory condition, an attempt should be made to improve the quality of the blood, for as long as anæmia persists, the disease is seldom amenable to treatment. The tartrate, carbonate and ammonio-citrate of iron are to be preferred to other drugs. The medicine should be administered after the meals, and I have often found that the addition of a few minims of the tincture of belladonna is extremely beneficial in preventing a recurrence of the abdominal attacks.

Cod-liver-oil is seldom of much use in the early stages of the disorder, but as soon as the pain has subsided it may be cautiously employed, and the dose gradually increased. If the crude oil produces nausea, malt extract with cod-liver-oil (Kepler) may be substituted for it with advantage.

When the symptoms of gastric catarrh present themselves, the child should be kept indoors, and, if necessary, be confined to bed. The diet should consist entirely of milk, milk and soda-water, beef-tea, thin soup, or beef essence. Small doses of mercury and chalk may be administered every evening for a few days, followed the next morning by a saline purge. At a later period an alkaline mixture, with or without rhubarb or taraxacum, may be given once or twice during the course of the twenty-four hours.

If the digestion remains in a feeble state after the attack has passed away, the treatment appropriate for this condition must be employed (Chapter VIII.).

CHAPTER XI.

PAROXYSMAL HYPERACIDITY.

Acid dyspepsia arising from an excessive secretion of gastric juice such as occurs in adult life is seldom encountered below the age of puberty. Intermittent hyperacidity, on the other hand, does not appear to be an uncommon phenomenon in childhood. Thus, in some children the administration of a few grains of blue pill is invariably followed within a short time by pyrosis and a burning pain in the epigastrium, while in others indulgence in certain articles of diet, such as calf's liver, salt bacon, tea, and honey, immediately gives rise to an abnormal secretion of hydrochloric acid. I have also observed a similar disorder of the stomach to accompany the establishment of the catamenia, and in one case which came under my care for incontinence of urine the girl vomited large quantities of fluid containing about 0·3 per cent. of free hydrochloric acid during the two days prior to each menstrual epoch. Another girl, seven years of age, who attended my out-patient department at the Evelina Hospital for renal colic on the right side, and who had on two occasions voided a uric acid stone, often vomited fluid containing 0·27 to 0·35 per cent. of free hydrochloric acid coincidentally with each paroxysm. But in addition to these cases, which may be regarded as examples of reflex stimulation of the stomach, I have observed three instances where an abnormally acid gastric secretion recurred from time to time, and was accompanied by cerebral symptoms similar in character to those of migraine. Although there is reason to believe that the disorder in question is not a primary affection of the stomach, it nevertheless presents so many points of interest connected with the organs of digestion that I shall describe it under the title of 'paroxysmal hyperacidity.'

SKETCH OF A CASE.

A child from four to ten years of age is suddenly attacked during the course of the afternoon with severe headache. At first the pain chiefly affects the frontal or occipital region, but after a short time it tends to become diffused over the entire cranium. Any attempt to stand upright, to move the head, or even to swallow or cough, increases the pain, and during the height of the attack the patient often cries out at intervals as though he was suffering from meningitis, or becomes partially aphasic and unable to express his sensations in a coherent manner. After a variable interval the child complains of severe pain in the pit of the stomach, and draws up its knees and clasps the hands over the abdomen in order to obtain relief. As soon as the gastric symptoms have attained a certain degree of intensity, the headache usually abates, but is apt to return from time to time. Flatulent distension of the abdomen, with occasional eructations of gas, now makes its appearance, and pyrosis accompanied by a scalding pain behind the lower end of the sternum is experienced. Nausea and giddiness soon supervene, and the child suddenly sits up and empties its stomach without apparent effort. The act of emesis is followed by pain in the throat and choking, and the patient will endeavour to clear its mouth by continually spitting, or will cry out for water with which to wash away a sour taste. In some attacks the act of emesis is followed at once by the disappearance both of the headache and the gastric pain, so that the child will fall asleep and awake after a few hours quite restored to health. In other instances vomiting persists for some hours, and convalescence may not be established for several days. During the crisis of the pain, the face and extremities are cold, and the child not infrequently shivers and expresses great fear of being left alone. The pulse is small and usually slower than normal, and the temperature in the rectum is depressed. Examination shows that the total acidity of the vomit is unduly high, owing to the presence of an excess of free hydrochloric acid. The particles of food present in it exhibit signs of digestion, and the fluid, after filtration, rapidly dissolves albumin. At the termination of the attack a large quantity of pale urine is often voided. After the disorder has subsided, the secretion of hydrochloric acid is found to be normal.

In each of my three cases the complaint showed itself for the first time about the age of four. In one instance the mother stated that she had been very 'hysterical' before the birth of the child, but I was never able to elicit any history which pointed to the existence of nervous disease in the family. As a rule, the attack is immediately preceded by a period of unusually good health and spirits, and in one case the mother informed me that whenever her little son appeared to be particularly vivacious she at once put him to bed, with the view of warding off the 'bilious attack which was brewing.' At other times, general malaise, with want of appetite and constipation, is complained of for several days before the headache begins. Mental fatigue often appears to induce an attack, and in one of my cases it was necessary to stop the child's education for several months, owing to the frequent recurrence of the disease when he went to school. Physical exhaustion and severe mental emotions also seem to excite the disorder.

The initial symptoms of an attack usually commence soon after the mid-day meal, at which the child often exhibits an abnormally hearty appetite ; but occasionally the headache supervenes during the early hours of the morning, and prevents the patient from sleeping after three or four o'clock.

Although pain in the head is usually the first symptom to attract attention, it sometimes happens that an attack passes off without any headache worthy of the name. This is chiefly to be observed when the disorder recurs at short intervals, and runs its course within a few hours. At other times the headache only appears after the epigastric pain has persisted for some time, while occasionally it is replaced by a sense of extreme giddiness or dimness of vision. Lastly, it may be mentioned that in some attacks numbness and tingling of the fingers, or anæsthesia along the outer sides of the thighs and feet, precede or accompany the cerebral phenomena.

The acidity of the gastric contents varies in different attacks, and appears to bear an intimate relation to the severity of the cerebral symptoms and to the state of the stomach. Thus, when the attack commences after the mid-day meal, the vomit often exhibits an acidity equal to 0·45 to 0·52 per cent. HCl, the amount of free acid being also considerably in excess of the normal. When, however, vomiting ensues in the early morning, the total acidity may not exceed 0·38 per cent. HCl. Lactic acid can often be recognised in the ejecta after the mid-day meal. In severe cases the acidity of the vomit diminishes gradually with each act of emesis, so that towards the end of an attack the ejecta may consist entirely of alkaline and bile-stained mucus.

Flatulent eructations frequently precede the act of vomiting, more especially when the stomach contains food. The bowels are usually confined, but occasionally a sharp attack of diarrhœa appears to replace the vomiting, and exerts the same beneficial influence upon the symptoms. As long as the disorder persists, the appetite remains in abeyance, but thirst may be severe. It is an interesting fact that the administration of tepid water often relieves both the headache and the epigastric pain, and in one of my cases the nurse had learnt to make the child drink a tumblerful of water or milk directly the headache began, as the most certain means of affording relief. When, however, the complaint has already existed for some time, the relief obtained in this manner is only temporary, and the symptoms do not disappear until vomiting has taken place. As a rule, the temperature of the body is depressed at the commencement of an attack, but occasionally the thermometer in the rectum may register

100° or 101° F. for a few hours. The disorder usually runs its course in five to eight hours, but sometimes several days may elapse before the child regains its former state of health. The shortest attacks are those which occur after meals, and where the acidity of the gastric contents rapidly diminishes. When the disorder commences in the early morning, acute catarrh of the stomach not infrequently results from irritation of the abnormally acid secretion, and may persist for a day or two.

In the literature which deals with the subject of hypersecretion of the gastric juice, I have found several observations which appear to possess an important bearing upon the cases which have just been described. Under the title of 'gastroxynsis' Rossbach[1] has related several instances where the symptoms of migraine were associated with the secretion of an excessive amount of hydrochloric acid. In many of these cases the headache was accompanied by epigastric pain, and the writer appears to consider that the cerebral symptoms were due to the disordered condition of the stomach. Lépine[2] has recorded several similar cases under the title of 'gastroxie'; while Rosenthal[3] has invented the term 'vomitus hyperacidus' for the vomiting of an abnormally acid gastric juice which he had observed in school-children between the ages of seven and eleven. Lastly, an interesting case has recently been published by Snow,[4] in which an infant at the age of nineteen months was seized with convulsions, and vomited a large quantity of acid fluid, which gave a well-marked reaction with Gunzburg's solution. The temperature ranged between 100° and 102° F. during the five days that the attack lasted. The disorder recurred at intervals up to the age of five, and was finally cured by strict dieting and physical exercises. In this case, however, there appears to have been an absence of headache. In the published report of the discussion which followed the reading of this case, several gentlemen appear to have expressed their intimate acquaintance with the complaint, though it does not seem that they had tested the vomit for free hydrochloric acid; while the author himself only mentions that he obtained a positive reaction with phloroglucine-vanillin, but gives no details of any quantitative analysis. Since the main point of distinction between the cases I have described and those of recurrent catarrh of the stomach lies in the character of the gastric secretion, it is easy to mistake one complaint for the other, unless a careful examination be made of the vomit in every instance.

---

[1] *Deut. Arch. f. Klin. Med.*, 1884, s. 383.

[2] *Soc. Méd. des Hôpitaux*, 1885, p. 134.

[3] *Berl. Klin. Woch.*, 1887, s. 505.

[4] *Archiv. f. Pediat.*, 1893, p. 986.

ETIOLOGY.

Rossbach has suggested that in cases of acid vomiting the symptoms are due to a vaso-motor disturbance in the medulla, the effect of which is to produce headache and a secretion of abnormally acid gastric juice. This latter in its turn causes irritation of the mucous membrane of the stomach, and finally gives rise to vomiting. If this explanation is accepted, the disorder must be regarded as similar in its nature to migraine and other paroxysmal neuroses. It is probable that future investigations will show that gastroxynsis, gastroxie, vomitus acidus, and the cases which have just been described, are either identical or at most varieties of the same disease.

DIAGNOSIS.

Unless the vomit is submitted to a chemical examination, paroxysmal hyperacidity may easily be confounded with migraine or recurrent catarrh of the stomach. It is to be observed, however, that migraine usually occurs at a somewhat later period of life, and that its onset is preceded by ocular phenomena which are wanting in the other disorder. The headache, also, is localized to the frontal region, rather than diffused over the vault of the skull, and is never accompanied by severe pain at the epigastrium; while nausea invariably precedes the act of vomiting, and the ejecta do not exhibit any excess of hydrochloric acid. In cases of gastric catarrh, the headache is much less severe than in cases of hyperacidity. Nausea is a constant symptom, and retching and vomiting recur at short intervals, while the ejecta consist of mucus tinged with bile. The temperature is subnormal, the bowels are confined, and the tongue is thickly coated.

TREATMENT.

All conditions which are apt to excite an attack must be avoided as far as possible, and if the disorder shows a tendency to recur frequently, the child should be kept from school and allowed to indulge in plenty of exercise in the fresh air. The meals must be taken at regular intervals, and the food should be of a kind which is easily digestible. If the bowels are confined, they should be regulated by means of some simple laxative, such as cascara, sulphur, senna, or compound liquorice powder. At the commencement of an attack, the child should be put to bed and the room darkened. In many cases, an emetic of ipecacuanha wine or sulphate of zinc appears to relieve the headache more quickly than anything else, while if the epigastric pain is severe, a tumblerful of hot water may be administered. Occasionally fifteen grains of antipyrin, or six grains of phenacetin appear to cut short an attack when given at an early stage. As soon as vomiting has taken place, the child should be encouraged to drink plenty of warm water, rendered slightly alkaline with carbonate of sodium with the view of soothing the gastric mucous membrane; or it may be allowed to suck a small piece of ice from time to time. In those cases where catarrh of the stomach ensues from irritation of the acid secretion, and the patient retches incessantly, an enema containing chloral and bromide of potassium may be administered. Among the various remedies I have tried for the purpose of preventing a recurrence of the disease, bromide of sodium combined with belladonna, or the solution of nitroglycerine, have proved of most value.

CHAPTER XII.

DYSPEPSIA DEPENDENT UPON DISEASE OF OTHER ORGANS.

PHTHISIS.

CHILDREN who are the subjects of tubercular disease of the lung are very liable to exhibit symptoms of deranged digestion. In cases of acute miliary tuberculosis, vomiting and diarrhœa are frequently present, and are often so severe as to exert a notable influence upon the course of the disease. In such cases it is usual to find follicular ulceration of the stomach and colon after death along with severe catarrh of the mucous membrane of the digestive tract. It is, however, in chronic phthisis that the symptomatic dyspepsia is of greatest importance, for in many cases the pain and vomiting which ensue after food completely mask the pulmonary complaint, and may lead to a serious error in diagnosis.[1] The dyspepsia varies in character at different periods of the lung disease, so that it is possible to draw a clinical distinction between the dyspepsia of early phthisis and that which accompanies the terminal stage of the tubercular complaint.

---

[1] Author, 'Dyspepsia of Phthisis,' 1894, p. 104.

The Initial Dyspepsia.—Nearly 70 per cent. of all cases of phthisis exhibit symptoms of dyspepsia at the commencement of the lung disease. The complaint shows itself more frequently in girls than boys, and is especially common in those who possess an hereditary tendency to tuberculosis. It is always more severe in chronic cases than in those which pursue an acute course. *Pain* at the epigastrium during the process of digestion is present in every case. At first the symptom only occurs at intervals, but after a time it is experienced after each meal. In some cases the epigastric discomfort is described as a sense of weight or fulness, and is not infrequently referred to the chest rather than to the abdomen; in other instances it is of a scalding character, and is diffused over the whole of the epigastric region and between the shoulders. As a rule, the pain subsides spontaneously before the next meal, but occasionally it may persist all day, being temporarily relieved by the ingestion of food. *Vomiting* is a frequent symptom in the early stage of pulmonary tuberculosis, and usually shows itself in the first instance when the patient rises from bed in the morning. It is invariably preceded by tickling in the throat and severe cough. Retching follows directly upon a violent expiratory effort which is necessary to dislodge and expel a small quantity of sticky mucus from the pharynx or bronchial tubes, and if the stomach happens to contain any mucus, this is rejected at the same time. Occasionally, when the retching proves severe, a small quantity of bilious or blood-stained fluid may be vomited. It is to be noticed that the attack is neither preceded nor accompanied by nausea, giddiness nor faintness, and possesses no tendency to spontaneous recurrence, while the child is often able to eat its breakfast without further discomfort.

In other instances, or at a later stage in the same case, another variety of vomiting may be observed. At first sight this seems to be directly excited by the ingestion of food, and takes place most frequently after the evening meal. Careful examination, however, usually shows that in this case, also, the vomiting is preceded and caused by cough. The attacks are apt to be accompanied by pallor of the face and palpitation, and the patient will often decline to proceed with the meal. In severe cases every attempt to partake of food is followed by an attack of coughing, which terminates in emesis, and the child fears to indulge its appetite on account of the discomfort which invariably follows.

Disorders of the Appetite.—In the early stages of the complaint the appetite may present little or no deviation from the normal, but as the disease progresses it tends to diminish, and may even disappear.

Sometimes the patient seems to be ravenously hungry for several days in succession, and after eating a heavy meal will still declare himself to be unsatisfied. More commonly the desire for food varies from day to day and from meal to meal, or a good appetite will vanish suddenly after slight indulgence. The subjects of this form of dyspepsia often exhibit a remarkable repugnance to certain articles of diet, notably to meat-fat. This peculiarity exists to a greater or less degree in almost every case, and in a large proportion only shows itself for the first time at the commencement of the pulmonary disease. In many cases saccharine substances also prove distasteful, from the fact that they give rise to acidity or flatulence. On the other hand, many patients exhibit an inordinate affection for sour or acid substances, like lemons and vinegar.

Reflex Cough is another common symptom of early phthisis. Soon after a meal, especially if the food which has been taken happens to be difficult of digestion, the child is seized with a severe attack of coughing, which often terminates in the expulsion of the contents of the stomach. Sometimes the cough comes on without any warning, but usually it is preceded by a sense of irritation at the back of the throat or base of the tongue. This phenomenon is probably of a reflex nature, and arises from irritation of the nerves of the pharynx or stomach by the food.

Flatulence frequently occurs after a meal, and in many cases the epigastric discomfort which is experienced during the course of digestion is due to this cause. The *bowels* are almost invariably confined, and the stools are pale, fœtid, and largely mixed with mucus. The *tongue* is large, flabby, and indented along its margins by the teeth. The salivary secretion is acid, and rapid rotting of the *teeth* is frequently observed. Some patients suffer from extreme *thirst*, but this symptom is not always a noticeable feature of the complaint. *Anæmia* is always present, and the severity of the dyspepsia usually varies directly with the deficiency of hæmoglobin. Physical examination of the stomach in these cases seldom demonstrates the existence of any enlargement of the organ, but if air is introduced into it by means of a bellows, the stomach can be ballooned with ease. The secretion of hydrochloric acid usually diminishes as the complaint progresses, more especially if the pulmonary disease is accompanied by high fever. In those cases where the phthisis becomes arrested at an early stage, the symptoms of indigestion disappear spontaneously as the general health improves; but if the pulmonary mischief is progressive, the gastric disorder gradually alters in character, and becomes more severe.

The Terminal Dyspepsia.—Coincidently with the development of cavities in the lung, the mucous membrane of the digestive tract is attacked by a chronic form of inflammation. In the stomach this morbid process commences in the connective tissue between the gastric glands, and by the gradual production of fibrous tissue gives rise to a diffuse cirrhosis of the organ, with destruction of its secretory structures. The duodenum and the rest of the intestine are affected in a similar manner, so that eventually the secretory and absorptive powers of the digestive canal are reduced to a minimum. Occasionally the pancreas and the salivary glands also exhibit signs of cirrhosis.

Painful sensations at the epigastrium, either occurring spontaneously or as the result of an effort of digestion, are comparatively rarely encountered in this form of dyspepsia. When pain does occur, it is seldom severe, but may be increased by pressure over the region of the stomach. Vomiting is only an occasional symptom, and usually takes place in the early morning or after a meal. The vomited material consists of undigested food mixed with a large quantity of acid mucus. The bowels are generally irregular in their action at the commencement of the complaint, periods of constipation alternating with sharp attacks of diarrhœa. Towards the last, however, the bowels are relieved every hour or two, and the exhaustion which ensues from this diarrhœa materially hastens the fatal termination. In the early stages the tongue is redder than normal, and presents a bright-red tip and a dorsum covered in a patchy manner with yellow fur. Later on the surface acquires a morbidly red and shining appearance, or it becomes dry in the centre and presents aphthous sores along its edges. The appetite remains in abeyance, but severe thirst is a constant source of complaint.

Physical examination of the abdomen almost always reveals some degree of dilatation of the stomach, with tenderness on deep pressure over the region of the epigastrium. The secretion of hydrochloric acid steadily diminishes, until free acid is altogether wanting in the contents of the organ. When the mucous membrane has been permanently and severely damaged by the inflammatory process, the peptic and rennet ferments also fail.

Treatment.

The general treatment of the disorder must be subservient to that of the pulmonary disease. In the selection of an appropriate dietary, the capricious tastes which characterize this form of dyspepsia should be borne in mind, and all greasy and saccharine articles prohibited. In other respects the complaint may be treated on the same lines as those laid down for cases of weak digestion (p. 272). Arsenic is of considerable value in mild cases, where the appetite is deficient, and is most advantageously prescribed in combination with dilute hydrochloric acid and infusion of gentian. When anæmia is a conspicuous feature of the case, the administration of iron is usually attended by success; but this drug should be replaced from time to time by a mixture containing bicarbonate of sodium, rhubarb and taraxacum, while a few grains of gray powder or blue pill may be given once or twice a week at night-time. For the purpose of regulating the bowels, cascara, sulphur, or some other laxative, should be employed.

Morning sickness is best treated by the use of gargles containing chlorate of potassium and borax, or by the administration of a dose of linctus or codeia jelly. When the vomiting ensues as a result of the reflex cough excited by the introduction of food into the stomach, the chief reliance must be placed in sedatives. Of these, morphine, nepenthe and hydrocyanic acid are the most useful, and should be given about ten minutes before the commencement of each meal. In some cases the application of a solution of cocaine to the pharynx immediately before each meal will prevent emesis. A small quantity of diffusible stimulant is also a valuable adjunct to the other methods of treatment.

The terminal dyspepsia of phthisis does not usually require any active treatment. If, however, vomiting is a troublesome symptom, recourse must be had to bismuth and morphine, or to other remedies which are appropriate to the treatment of chronic gastro-intestinal catarrh.

Children suffering from acute *pneumonia* occasionally present gastric symptoms of an acute character arising from catarrh of the stomach and duodenum. These phenomena have already been discussed in Chapter VI.

In cases of *bronchiectasis* the stomach and intestine are apt to become affected with a form of chronic catarrh which in every way resembles that of the terminal stage of phthisis.

CARDIAC DISEASE.

Children who suffer from valvular disease of the heart are apt to develop symptoms of gastric and intestinal dyspepsia when compensation fails, and in some instances the disorder of digestion is of such importance as to exert a deleterious influence upon the course of the original malady.

Pathology.

When the stomach is opened after death, the mucous membrane is found to be swollen and purple in colour from extreme congestion. In some cases the organ contains a considerable quantity of bilious fluid, while in others the contents consist of thick mucus, which may be tinged with blood. In long-standing cases the mucous membrane in the fundus is often pigmented, but when death has occurred rapidly, numerous punctiform hæmorrhages may usually be discerned scattered over the cardiac and middle zones of the viscus. Sometimes actual erosion ensues from this cause. The duodenum and large intestine present similar appearances. When examined with the microscope, the gastric mucous membrane is found to be considerably swollen, and the capillary vessels which ramify beneath the superficial epithelium and in the interstitial tissue are engorged with blood. The basement membrane of the gastric glands is thickened, and the secretory epithelium appears swollen and granular, or in a state of active proliferation. The ducts of the tubules are usually blocked with mucus, detached epithelium, and blood-cells. In the pyloric region the signs of chronic congestion are less pronounced, but it is not unusual to find a considerable amount of round-cell exudation between the glands, which in chronic cases may have undergone partial organization. The submucous coat of the stomach exhibits engorgement of its vessels, with a large number of deeply-staining cells disseminated through its connective tissue.

Symptoms.

The first symptom to attract attention is usually distension of the abdomen after meals, accompanied by flatulence and dyspnœa. Occasionally the symptoms occur with alarming suddenness, and give rise to cyanosis of the face and lips. They usually subside after the expulsion of gas from the stomach, but fatal syncope may result from embarrassment of the heart's action.

Sooner or later vomiting makes its appearance, and is especially apt to occur after the last meal of the day. The food which is rejected presents no appearance of digestion, and is usually mixed with an excess of mucus. In advanced cases vomiting occurs after every meal, and even a small quantity of water may strictly regulated, to the exclusion of excess of fluid and starchy substances. In severe cases the food may be peptonized. A small quantity of warm spirit and water taken with the meals often appears to aid digestion, and to prevent accumulation of gas in the stomach. The last meal ought to be taken early in the evening, and be composed of light and easily-digestible materials.

In the last stage, where the vomiting is incessant and the ejecta consists mainly of bile and mucus, an attempt must be made to preserve nutrition by means of enemata.

KIDNEY DISEASE.

Dyspepsia is a common symptom of Bright's disease. In some cases the nausea and vomiting which accompany the renal mischief appear to depend upon uræmic poisoning, but in the majority of cases the disordered state of the digestion arises from chronic catarrh of the stomach and intestine.

Pathology.

The aspect of the stomach after death from Bright's disease varies considerably in different cases. Occasionally the inner surface of the organ is covered with a thick layer of mucus, and when this has been removed, the mucous membrane presents a diffuse capilliform injection, with here and there a punctiform hæmorrhage or erosion. More commonly, the inner surface of the stomach appears pale and sodden, as though it had been long soaked in saline solution. In some cases actual œdema of the walls of the viscus exists, but this I have never observed except in cases where general anasarca was present. Pigmentation of the mucous membrane is chiefly encountered in cases where the death has ensued from gradual failure of the heart. The intestines seldom exhibit any obvious signs of disease to the naked eye, but occasionally the mucous membrane of the ileum in the vicinity of the ileo-cæcal valve is found to be swollen and congested, while a few ulcers exist in the colon and sigmoid flexure. The microscopic appearances of the stomach vary according to the nature of the renal disease. Thus, in four cases of acute parenchymatous nephritis, I found that the gastric glands presented unmistakable signs of acute catarrh, while in one instance, where death had resulted from chronic inflammation of the tubules of the kidney, the glands of the stomach exhibited the appearances which have already been described as indicative of chronic gastric catarrh (Fig. 14). Up to the present time I have not had an opportunity of examining the stomach in a case of chronic interstitial nephritis in a child, but if any conclusion may be drawn from the morbid condition of the stomach which accompanies the renal affection in adult life, it is probable that interstitial gastritis will be found to be the principal feature of the complaint.

Etiology.

Gastric catarrh occurring during the course of Bright's disease appears to arise from the retention of certain excrementitious products within the system. It is known that the mucous membrane of the gastro-intestinal tract can excrete urea after it has been injected beneath the skin of an animal, but the elimination of the pure substance fails to excite acute catarrh.[1] On the other hand, the subcutaneous administration of many of the salts of iron, arsenic, antimony, etc., is followed at once by the appearance of these drugs in the stomach, and by acute gastritis. When sulphate of iron has been employed, the salt can be detected in the substance of the peptic cells after the tissue has been treated with ferrocyanide of potassium. In several cases of acute nephritis I evaporated the urine to a small bulk, and then treated it with an excess of absolute alcohol. The resultant precipitate was extracted with distilled water, evaporated, and again precipitated with alcohol. Finally a brownish-red, deliquescent substance of an albuminous nature was obtained, which, when injected into rabbits in the proportion of 0·2 gramme per kilo of the body-weight, gave rise to severe dyspnœa and fatal convulsions. At the necropsy severe congestion of the stomach and intestines was found, with numerous hæmorrhages in the mucous membrane. The lungs were œdematous, and punctiform hæmorrhages were often present beneath the visceral layer of the pleura. Although the excretion of urea does not of itself produce catarrh of the gastro-intestinal tract, it is extremely probable that some other product of metabolism which is eliminated with it may be responsible for the inflammatory lesion.

---

[1] Author, *Virchow's Archivs*, vol. cxviii., s. 187.

Symptoms.

The chief symptoms of the gastritis of renal disease consist of nausea, vomiting, loss of appetite, thirst, and headache. The sensation of sickness is chiefly experienced in the early morning, and is at first capable of being relieved by food and stimulants. In severe cases the nausea may persist all day, and is apt to be accompanied by salivation and a constant saline taste in the mouth. Two varieties of vomiting may be observed, one of which is central in origin, and may be termed 'uræmic,' while the other arises directly from the inflammatory state of the stomach. The former chiefly occurs in cases of acute Bright's disease, where the secretion of urine is greatly reduced, or when a subacute attack supervenes upon the chronic disease. Under these conditions the child immediately rejects everything it swallows, and is liable to severe and sudden attacks of retching between meals. Nausea is always present, and in most cases headache and dimness of vision accompany the attack. When vomiting arises from chronic gastric catarrh, it usually occurs in the early morning before breakfast, and has for its object the expulsion from the stomach of a considerable quantity of alkaline mucus. Urea can usually be recognised in the contents of the stomach, and in one case where I had the organ evacuated every morning, it sometimes amounted to more than 0·7 per cent. Occasionally the alkalinity of the mucus is due to the conversion of a portion of the urea into carbonate of ammonium. Many children also suffer from vomiting after the evening meal. Anorexia is a constant symptom in these cases, and the utmost loathing may be expressed towards every kind of food. Thirst is usually excessive, and the child often exhibits a marked preference for acid fluids. In chronic cases these symptoms are apt to subside spontaneously from time to time, and their remission frequently coincides with an increased elimination of urine. The bowels are usually confined, but occasionally attacks of diarrhœa occur, accompanied by fœtid or ammoniacal stools.

Treatment.

When vomiting is severe and the child exhibits symptoms of exhaustion, an enema containing chloral and bromide of potassium is often of considerable service. In cases of uræmic vomiting associated with parenchymatous nephritis, one-fifteenth of a grain of morphine combined with one-hundredth of a grain of sulphate of atropine may be injected beneath the skin with advantage. At the same time the bowels should be purged with jalap or scammony, and a hot-air bath be given if possible. In the chronic forms of the complaint, a full dose of Carlsbad salts may be given each morning, and in obstinate cases lavage of the stomach can be undertaken. If the appetite is deficient, dilute phosphoric acid with infusion of gentian should be administered between the meals, while the process of digestion may be aided by the employment of pepsin and hydrochloric acid after food.

NERVOUS DISEASE.

Almost every disease of the central nervous system, whether acute or chronic, is accompanied by symptoms of disordered digestion, which in some cases attains such a degree of importance as to distract attention from the primary complaint.

Pathology.

Examination of the stomach after death seldom demonstrates the existence of any morbid change other than an extreme degree of post-mortem digestion. Thus, in fourteen cases of meningitis, cerebral tumour, or abscess, accompanied by severe vomiting, where I examined the stomach and intestine after death, the microscope failed to detect any sign of catarrh or other morbid condition.

Physiology.

As a rule the secretion of the stomach remains unaffected in cases of organic disease of the brain and spinal cord. In two cases of tubercular tumour of the brain, however, the gastric contents exhibited an abnormal degree of acidity, the total amount of free and combined hydrochloric acid varying from 0·25 to 0·32 per cent. In these cases, also, the motor activity of the stomach appeared to be somewhat exaggerated, and the organ was found to be free from food two hours after a meal.

Symptoms.

Vomiting is a constant and important symptom of cerebral disease in childhood. It usually commences at an early stage of the complaint, and persists until paralysis has set in. As a rule, the emesis takes place immediately after the ingestion of food, the contents of the stomach being pumped up without effort, and usually without any antecedent feeling of nausea. At other times severe retching occurs between the meals, and terminates in the rejection of a small quantity of bilious fluid. The bowels are usually confined, and the pulse slow and markedly irregular.

Treatment.

In severe cases nutrition must be maintained by means of nutrient enemata, while an attempt is made to control the vomiting by means of morphine or rectal injections containing chloral and bromide of potassium.

APPENDIX.

CHEMICAL EXAMINATION OF THE CONTENTS OF THE STOMACH.

IN order to obtain results which are capable of comparison in different cases, three points require attention. In the first place, the test meal must be identical both in quantity and quality in each instance; secondly, the contents of the stomach must be evacuated after a fixed interval of time; thirdly, the same methods must be employed in the detection and estimation of the products of digestion.

The Test Meal.

In the case of infants, an ounce of cow's milk mixed with an equal quantity of water should be administered when the stomach is empty, and the residue withdrawn at the end of an hour. If chronic gastric catarrh is present, the organ should be washed out with warm water before the milk is given. In older children, a piece of dry bread of definite weight, lightly dusted over with white sugar, may be given along with a small cupful of weak tea and milk. The stomach should be emptied at the end of an hour and a half. As a rule, a soft catheter of sufficient diameter is all that is required to procure the evacuation of the gastric contents, but should any difficulty be experienced, the process may be aided by suction with a glass syringe.

Character of the Gastric Contents.

The quantity which can be extracted from the stomach varies in different cases, but as a rule it exceeds ten cubic centimetres in an infant, and more than twice that amount in older children. In normal cases the material is semi-fluid in consistency, and possesses an odour resembling that of fresh meat; but when fermentation has taken place in the stomach, the smell is often sour, rancid, or pungent.

Filtration.

This is performed by means of a glass funnel and a piece of porous paper. The rapidity of the process varies with the character of the material, being slow and imperfect when much mucus and soluble albumin is present, but comparatively rapid when the mixture contains free hydrochloric acid. The appearance of the residue upon the filter affords a general indication of the activity of the gastric juice. If it is considerable in quantity, and the particles of food present only slight evidences of digestion, the gastric secretion is probably deficient; but if only a small amount of curdled milk or bread remains, or the pieces exhibit a ragged and gelatinous appearance, the gastric secretion is probably active. Excess of mucus is shown by a glairy or sticky condition of the residue, and by the presence of opaque pellets upon the filter. Microscopical examination of the residue reveals a variable amount of fat, casein, starch granules, epithelial cells, and micro-organisms.

The *filtrate* is a limpid, yellow, and slightly opalescent fluid, which possesses an odour similar to that of the crude extract. If free hydrochloric acid is present, it resists decomposition for a considerable time; but if the mineral acid is absent, the fluid soon undergoes putrefaction. It is usually acid in reaction, but under certain circumstances it may be neutral, or even alkaline. The acidity may be due either to hydrochloric acid, to acid salts, or to organic acids arising from fermentation of the food.

To Detect Free Hydrochloric Acid.

The best test for this purpose is the solution devised by Gunsburg, which consists of phloroglucine, two parts; vanillin, one part; absolute alcohol, thirty parts. A few drops of this solution are mixed with an equal quantity of the filtrate in a porcelain dish, and gently heated over the flame of a spirit-lamp. The presence of free hydrochloric acid is denoted by the appearance of a rose colour upon the dish along the line of evaporation.

To Detect the Organic Acids.

1. *Lactic Acid.*—This acid is produced by the fermentation of lactose and other carbo-hydrates. It can be readily recognised in the filtrate by means of a solution composed of carbolic acid, ten cubic centimetres; tincture of perchloride of iron, one drop; distilled water, twenty cubic centimetres. The mixture possesses an amethyst-blue colour, which changes to canary-yellow upon the addition of a trace of lactic acid (Ueffelmann).

2. *Butyric Acid.*—This is a product of lactic fermentation. It can be recognised in the filtrate by its peculiar rancid smell, and by the fact that with Ueffelmann's solution it produces a dirty-brown precipitate.

3. *Acetic Acid* is occasionally formed in the stomach by the fermentation of alcohol. It may be recognised by its pungent smell, and also by the red coloration produced by the addition of perchloride of iron to the filtrate, which has been neutralized with caustic soda.

To Estimate the Total Acidity.

Ten cubic centimetres of the filtered contents of the stomach are mixed in a porcelain dish with a few drops of an alcoholic solution of phenolphthallin. A Mohr's burette is then filled with the decinormal solution of soda, and the fluid is allowed to fall drop by drop into the porcelain dish, the contents of which are continually stirred with a glass rod. The appearance of a faint rose colour, which does not disappear upon agitation of the fluid, indicates the exact point of neutralization. The stopcock of the burette is then turned, and the number of cubic centimetres of the soda solution which have been employed is read off upon the scale and noted. Each cubic centimetre of the decinormal solution of soda requires 0·00365 gramme of hydrochloric acid to effect its neutralization, so that, in order to calculate the amount of the acid present in the ten cubic centimetres of the filtrate, it is only necessary to multiply 0·00365 by the number of cubic centimetres of the soda solution which were used. Thus, if seven cubic centimetres of the decinormal solution of soda were required, it is evident that the ten cubic centimetres of the filtrate contained 0·00365 × 7 = 0·02555 gramme of the acid, or, in other words, the acidity of the filtrate was equivalent to 0·2555 per cent. HCl. It is sometimes preferable to express the result in terms of the soda solution, in which case the total acidity in the above-mentioned case would be represented by the number 70, since it was found that seventy cubic centimetres of the alkaline solution would be required to effect the neutralization of one hundred cubic centimetres of the acid filtrate.

Quantitative Analysis.

Hydrochloric Acid.—This acid occurs in the contents of the stomach in two forms—either in a free state, or combined with the albuminous constituents of the food.

The free acid is most readily estimated by means of a method invented by Mintz, which is based upon the fact that, when caustic soda is added to a solution containing both the free and combined mineral acid, the whole of the former will be neutralized before the latter is affected.

Method.—Ten cubic centimetres of the filtrate are placed in a test-glass, and into it a decinormal solution of soda is allowed to fall drop by drop from a graduated burette. From time to time a drop of the mixture is transferred to a porcelain dish, and tested with the phloroglucine-vanillin solution. As soon as the reaction for the free acid fails to appear, it is evident that the whole of the free acid has been neutralized, and it is therefore only necessary to multiply the number of cubic centimetres of the soda solution which have been required for this purpose by 0·0365, to obtain the percentage amount of the free acid which was present in the contents of the stomach.

The *total quantity of hydrochloric acid* (free and combined) can be most readily estimated by the method proposed by Hehner and Seemann.

Method.—Ten cubic centimetres of the filtered contents of the stomach are exactly neutralized with the decinormal solution of soda, and the amount of the latter required for the purpose is carefully noted. By this means all the various acids present in the filtrate are converted into their corresponding salts of sodium (chlorides, lactates, butyrates, etc.). The mixture is now evaporated, and the residue calcined in a platinum crucible. The cinders, which consist solely of the carbonate and chloride of sodium, are then extracted with a small amount of distilled water, and to it is added a quantity of the decinormal solution of hydrochloric acid, equivalent to the decinormal soda solution which was originally used for the purpose of neutralization. A portion of this acid goes to neutralize the carbonates resulting from the oxidation of the organic salts, while the excess represents the amount of chlorides present in the solution, or, in other words, the total quantity of hydrochloric acid contained in the ten cubic centimetres of the filtrate. In order to estimate this latter, it is necessary once more to triturate with the standard solution of soda until neutralization is effected, when. by noting the number of cubic centimetres employed for this purpose, the percentage of the hydrochloric acid is easily calculated. Thus, supposing that four cubic centimetres of the soda solution are required for the final neutralization, then the ten cubic centimetres of the filtrate contained $4 \times {\cdot}00365 = 0{\cdot}0146$ HCl, or $0{\cdot}146$ per cent. HCl. This process estimates the total amount of the acid in the filtrate, so that, if it be desired to determine the amount of combined acid only, it is necessary to deduct from the result that which was obtained by employing the method of Mintz (free acid).

Lactic Acid.—The quantitative estimation of lactic acid is seldom of much importance in cases of disease in children. If necessary, however, it can be performed in the following way: Ten cubic centimetres of the filtrate are heated with a few drops of sulphuric acid in order to coagulate any albumin which may be present. After filtration, the liquid is evaporated over a water-bath until it attains a syrupy consistence; it is then mixed with ten cubic centimetres of distilled water and evaporated to dryness, in order to drive off the volatile acids. The residue is thoroughly extracted with an excess of ether, the ether separated and gently evaporated. The residue, which consists entirely of lactic acid, is dissolved in water and triturated with the decinormal solution of soda. The number of cubic centimetres of the alkaline solution required to affect neutralization is noted, and the percentage quantity of lactic acid is determined by multiplying this number by $0{\cdot}09$.

The Detection and Estimation of the Ferments.

Pepsin is always present in a gastric juice containing free hydrochloric acid, but its activity varies in different cases. The presence of the ferment is easily recognised by the action of the filtered contents of the stomach upon egg-albumin. About twenty cubic centimetres of the fluid are placed in a small flask along with one or two cubes of the albumin, and maintained for a couple of hours at a temperature of 38° C. If the filtrate contains no free acid, ten cubic centimetres of a 0·2 per cent. solution of hydrochloric acid should be added to it. The first indication of peptic action is the development of a translucent appearance of the albumin at its periphery, followed by the detachment of the substance in the form of fine flakes, which ultimately dissolve in the surrounding fluid. The time occupied by the solution of the cube affords a rough estimate of the activity of the ferment.

The rennet ferment is invariably present in the secretion of a child's stomach, and is the last to disappear in cases of gastric disease. It can be recognised in the contents of the stomach by the fact that after neutralization the filtrate coagulates milk, the rapidity with which this occurs varying directly with the quantity of the ferment present.

RECIPES REFERRED TO IN TEXT.

R. 1. *Barley Water.*—Mix two teaspoonfuls of pearl barley with a pint of cold water, and boil gently in a saucepan to two-thirds of its original volume; strain through fine muslin, and keep in a cool place. To be made fresh at least twice a day.

R. 2. *Oatmeal Water.*—Take one tablespoonful of the best oatmeal coarsely ground, add it to a pint of water, and simmer gently for one hour; strain, and add sufficient water to make a pint.

R. 3. *Oatmeal Jelly.*—Soak four ounces of coarse oatmeal in a quart of cold water for twelve hours; boil the mixture down to a pint, and strain through a fine cloth while still hot; as the mixture cools a jelly is formed; when required for use, mix with milk and warm.

R. 4. *Prepared Gelatine.*—Soak a teaspoonful of gelatine in half a pint of cold water for three hours; place the utensil in a saucepan of boiling water, and apply heat until the whole of the gelatine is dissolved; when the mixture cools it forms a transparent jelly, of which one teaspoonful may be added to each meal of milk. If isinglass is preferred, it may be prepared in a similar manner.

R. 5. *Lime Water.*—Take a piece of unslaked lime the size of a walnut, and place it in an earthen vessel with two quarts of filtered water; stir the mixture thoroughly, and allow it to stand; pour off the clear fluid when required for use.

R. 6. *Beef Tea.*—Cut up a pound of lean beef into minute pieces, and mix it with two pints of cold water; allow it to simmer for two hours, care being taken not to heat to the boiling-point.

R. 7. *Mutton Broth.*—Cut up a pound of lean mutton into small pieces, add a quart of cold water, and allow the mixture to simmer for three hours; remove scum from time to time; strain off the fluid, let it stand till it is cold, then remove the fat.

R. 8. *Veal Tea.*—Take a pound of minced veal free from fat, mix with a pint and a half of barley-water, and heat in a slow oven for three hours; strain and skim.

R. 9. *Chicken Tea.*—Cut up a fowl into small pieces; put it in an earthen vessel, with some salt and three pints of water; let it boil for three hours, then allow it to cool, and skim.

R. 10. *Flour Ball.*—Take a pound of Chapman's entire wheaten flour, enclose it in a pudding-cloth. and boil for ten hours; when cold, remove the outer layer of the mass and reduce the interior to powder; when required for use, one teaspoonful of the powder is carefully rubbed up with two tablespoonfuls of fresh-boiled milk, and afterwards mixed with five ounces of hot milk and barley-water. This preparation should not be given to infants less than six months old.

R. 11. *Barley Jelly.*—Boil five ounces of granulated barley in a quart of water until the volume of the mixture is reduced to a pint; strain through a fine cloth.

Bread Jelly.—Place four ounces of stale bread in a basin of cold water, and allow it to soak for seven hours; remove the pulp, and firmly squeeze out the water; boil gently for an hour and a half, strain, and allow the fluid to cool, when it will set as a fine jelly; when required for use, mix one tablespoonful with eight ounces of warm water, and add a sufficiency of milk or raw meat juice.

R. 12. *Raw Meat Juice.*—Finely mince a pound of best rump-steak free from fat; place it in an earthen vessel with sufficient cold water to cover it, and allow it to soak for three hours; express the juice through muslin. Two or three ounces may be given in the course of the twenty-four hours.

R. 13. *Scraped Meat Pulp.*—Take half a pound of the best rump steak, and scrape it with a knife until it is reduced to shreds. One teaspoonful or more may be given three times a day with a little sugar, or in the form of sandwiches, with thin bread-and-butter.

Raw Meat Pulp.—Take a pound of lean beef or mutton, cut it into small pieces, and reduce it to a pulp with a pestle and mortar; pass the pulp through a fine sieve. This preparation is less digestible than the former, since it contains a large amount of connective tissue.

R. 14. *Whey.*—To a pint of warm new milk add a teaspoonful of rennet (Benger); when the curd has formed, break it up with a fork, and allow it to subside; decant the clear whey and boil it; milk, cream, or brandy can be added as desired.

R. 15. *White Wine Whey.*—Boil a large cupful of new milk in a clean saucepan, and add a wineglassful of good sherry wine; boil for two minutes, strain off the curd, and add a little sugar; the proportion of the two ingredients must be varied according to the strength of the stimulant required.

R. 16. *Infusion of Malt.*—Three full tablespoonfuls of crushed malt are thoroughly well mixed in a jug with half a pint of cold water;

the mixture is allowed to stand for twelve hours; the supernatant liquor is then carefully decanted off from the sediment, and strained through two or three folds of muslin until it is fairly clear; the product is about six ounces, and is rich in maltose and diastase; it it prone to ferment, and must therefore be prepared fresh each day, or be preserved in a well-corked bottle by the addition of a few drops of chloroform. This latter can be easily dissipated by heat before the infusion is used. (Sir W. Roberts.)

CREAM MIXTURES MANUFACTURED ACCORDING TO DEFINITE FORMULÆ (ROTCH).

	Formula.	*Composition.*
1.	Fat, 1·00.	Cream, 2 ounces.
	Sugar, 5·00.	Milk, 2 ounces.
	Proteids, 0·75.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 7 drachms.
		Water, 15 ounces.
2.	Fat, 2·00.	Cream, 4 ounces.
	Sugar, 5·00.	Milk, none.
	Proteids, 0·75.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 7 drachms.
		Water, 15 ounces.
3.	Fat, 2·00.	Cream, 4 ounces.
	Sugar, 5·50.	Milk, 1½ ounces.
	Proteids, 1·00.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 8¾ drachms.
		Water, 13½ ounces.
4.	Fat, 2·50.	Cream, 5 ounces.
	Sugar, 6·00.	Milk, none.
	Proteids, 1·00.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 9 drachms.
		Water, 14 ounces.
5.	Fat, 3·50.	Cream, 7 ounces.
	Sugar, 6·50.	Milk, 1 ounce.
	Proteids, 1·50.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 9 drachms.
		Water, 11 ounces.
6.	Fat, 4·00.	Cream, 8 ounces.
	Sugar, 7·00.	Milk, none.
	Proteids, 1·50.	Lime-water, 1 ounce.
	Lime-water, 5·00.	Milk-sugar, 10 drachms.
		Water, 11 ounces.

Formula.	*Composition.*
7. Fat, 4·00. Sugar, 7·00. Proteids, 2·00. Lime-water, 5·00.	Cream, 8 ounces. Milk, 2½ ounces. Lime-water, 1 ounce. Milk-sugar, 9 drachms. Water, 8½ ounces.
8. Fat, 4·00. Sugar, 7·00. Proteids, 2·50. Lime-water, 5·00.	Cream, 8 ounces. Milk, 5 ounces. Lime-water, 1 ounce. Milk sugar, 8 drachms. Water, 6 ounces.
9. Fat, 4·00. Sugar, 7·00. Proteids, 3·00. Lime-water, 5·00.	Cream, 8 ounces. Milk, 7½ ounces. Lime-water, 1 ounce. Milk-sugar, 7 drachms. Water, 3½ ounces.
10.—*For Weaning.* Fat, 4·00. Sugar, 5·00. Proteids, 3·00. Lime-water, 5·00.	Cream, 8 ounces. Milk, 7½ ounces. Lime-water, 1 ounce. Milk-sugar, 3½ drachms. Water, 3½ ounces.

FORMULÆ REFERRED TO IN TEXT.

(*Doses suitable for an Infant Twelve Months Old.*)

F. 1. *Purgatives.*

Mag. sulph., gr. x.; mag. carb., gr. v.; sp. chlorof., min. iii.; glycerine, min. xv.; aq. menth. pip., ad ʒi. Three times a day.

Sod. sulph., gr. x.; quin. sulph., gr. ¼; tinct. nuc. vom., min. ½; acid. sulph. aromat., min. ii.; glycerine, ʒss.; aq. carui, ad ʒii. Every four hours.

Olei ricini, min. v.; mucil. tragac., min. xv.; syrupi, ʒss.; aq. menth. pip., ad ʒi. Every four hours.

Pulv. rhei, gr. i.; sodii bicarb., gr. ii.; sp. ammon. aromat., min. ii.; syr. zingib., min. x.; aq. cinnam., ad ʒi. Every four hours.

F. 2. *Intestinal Sedatives.*

Bism. carb., gr. iii.; pulv. cret. aromat., gr. iii.; pulv. ipec. co., gr. ¼; glycerine, min. xv.; sp. chlorof., min. iii.; aq., ad ʒi. Every four hours.

Pulv. ipec., gr. ⅛; pulv. cret. aromat., gr. iii.; bism. carb., gr. iii.; syrupi, min. x.; aq. chlorof., ad ʒi. Three times a day.

Tinct. opii, min. ½; acid. nit. dil., min. ii.ss.; glycerine, min. xv.; aq. chlorof., ad ʒi. Every four hours.

Pulv. hyd. c̄ creta., gr. ½; pulv. sacch. alb., gr. ii.; pulv. ipecac., gr. ⅙. Morning and evening.

Vin. antimon., min. ii.; tinct. opii, min. ½; aq. chlorof., ad ʒi. Every four hours.

F. 3. *Antiseptics.*

Resorcine, gr. iii.; bism. carb., gr. v.; sp. chlorof., min. ii.ss.; syrupi, min. x.; aq. cinnam., ad ʒi. Every four hours.

Glycerini acidi carbolici, min. i.; liq. calcis, ad ʒi. Every two hours.

Calomel, gr. ⅙; pulv. ipec. co., gr. i. Every four hours.

Creasoti, min. ⅙; tinct. iodi, min. ½; aq. camph., ad ʒi. Every hour.

F. 4. *Antispasmodics.*

Sod. bicarb., gr. i.ss.; sp. am. aromat., min. ii.ss.; sp. chlorof., min. i.; tinct. zingib., min. v.; glycerine, min. x.; aq. menth. pip., ad ʒi. Every four hours.

Mag. carb., gr. ½; sacc. alb., gr. iii.ss.; olei cajuputi, min. ⅙; sp. chlorof., min. i.; mucil. acaciæ, min. xx.; aq. anethi, ad ʒi. Every four hours.

Chloral hydras., gr. ½; pot. brom., gr. iii.; glycerine, min. x.; aq. anethi, ad ʒi. Every half-hour.

Pulv. rhei co., gr. iii.; sp. ammon. fœtid., min. iii.; sp. chlorof., min. iii.; tinct. card. co., min. xv.; aq. carui, ad ʒi. Every hour.

F. 5. *Liniments.*

Tinct. aloes, ʒi.; lin. saponis, ℥iii.; fiat. lin. To be rubbed over the abdomen along the course of the colon (E. Smith).

F. 6. *Artificial Digestives.*

Papain, gr. i.; sodii bicarb., gr. ii. Before each meal.

Papain, gr. ii.; sodii bicarb., gr. iii.; pulv. troch. menth. pip., gr. iii. After meals, three times a day.

Pepsin, gr. ii.; extr. casc. sag., gr. ½; extr. gent., q.s.; fiat. pil. Twice a day, after meals. For a child.

Pulv. ipecac., gr. ⅙; pulv. zingib., gr. i.; pulv. capsici, gr. ¼; extr. nucis vom., gr. ⅙; extr. gent., q.s. One pill twice a day, after meals. For a child.

F. 7. *Alteratives.*

Sodii bicarb., gr. i.; sp. ammon. aromat., min. ii.; syr. zingib., min. v.; tinct. rhei, min. v.; inf. gent. co., ad ʒi. Three times a day.

Acid. nit. dil., min. i.; acid. hydroch. dil., min. ii.; succus tarax., min. xv.; infusum rhei, min. x.; aq. chlorof., ad ʒi. Three times a day.

INDEX.

Acid, acetic, detection of, 362
——, ——, formation of, 362
——, butyric, detection of, 362
——, ——, formation of, 362
——, hydrochloric, detection of, 362
——, ——, estimation of, 363
——, ——, secretion of, 9
——, ——, ——, in acute gastric catarrh, 240
——, ——, ——, in chronic gastric catarrh, 191, 257
——, ——, ——, in dyspepsia, 74
——, ——, ——, in phthisis, 347
——, lactic, detection of, 362
——, ——, formation of, 362
——, ——, value of, 142
Acids, employment of, 40, 149, 247, 261
Acorn tea in diarrhœa, 207
Acute indigestion, 226
Acute gastric catarrh, 213
————, aphasia in, 229
————, apyrexial, 226, 229
————, diagnosis of, 241
————, etiology of, 213
————, epidemic, 217
————, febrile form of, 238
————, heredity of, 213
————, histology of, 220
————, membranous, 217, 219
————, morbid anatomy of, 218
————, primary, 214
————, recurrent, 233
————, secondary, 217
————, toxic, 236
————, treatment of, 242
————, varieties of, 225
Acute gastro-intestinal catarrh, 91
————, albuminuria in, 126
————, bacteriology of, 110
————, complications of, 123
————, diagnosis of, 128
————, diarrhœa in, 115
Acute gastro-intestinal catarrh—*Cont.*
————, etiology of, 92
————, general infection in, 112
————, histology of, 106
————, infective form of, 94
————, infection in, 99
————, morbid anatomy of, 100
————, onset of, 114
————, pain in, 115
————, prognosis of, 130
————, pulse in, 118
————, simple variety of, 92
————, symptoms of, 113
————, terminations of, 120
————, treatment of, 131
————, vomiting in, 114
Acute perforating gastric ulcer, 288
————, cases of, 288
Albuminuria in intestinal catarrh, 126, 183
Alimentary substances, digestibility of, 273
Analysis of condensed milks, 46
—— of cow's milk, 36
—— of human milk, 36
Anatomy of stomach, 1
Antimony in vomiting, 147
Antipyretics, 131
Antiseptics, administration of, 141
——, value of, 146, 210
Apoplexy in chronic intestinal catarrh, 186
Apparatus, the feeding, 51
——, care of, 52
Artificial feeding of infants, 33
Artificial human milk, 40
Asthma dyspepticum, 73, 230, 270
Ass's milk, 34
Athrepsia, 150
Atony of stomach, 75, 193
Atrophy of stomach and intestine, 164, 167
Auto-digestion of stomach, 103

Bacillus lactis aerogenes, 16, 111
Bacteria in milk, 113
—— of intestine, 16
—— of stomach, 14
Bacteriology of epidemic diarrhœa, 110
Bacterium coli commune in diarrhœa, 110
———— in intestinal catarrh, 217
Baginsky, 70, 97, 105, 112, 239
Ballard, 97, 98, 99
Barley-water, preparation of, 365
——, uses of, 39
Barlow, 290
Barthez, 153, 305, 313
Beaumont, 219
Bednar, 103
Benzoate of sodium, use of, 143
Benzol-naphthol, use of, 143
Besnier, 307
Beta-naphthol, use of, 143
Biedert, 69
Bignon, 307
Bile, secretion of, 14
Biliousness, 230
Billard, 283, 288, 312
Binz, 289
Bismuth in diarrhœa, 146, 210
——, salicylate of, 148, 210
Blood, condition of, in marasmus, 194
——, ——, in infancy, 193
Booker, 111, 112
Bottle, the feeding, 51
Bouchard, 143
Breast-feeding, 18
——, contra-indications to, 26
Breast, refusal of, 26
Breslau, 14
Brinton, 280
Bronchitis in acute intestinal catarrh, 125
—— in chronic intestinal catarrh, 185
—— in dyspepsia, 78
Burns, gastric ulcer in, 291, 297
Busch, 288
Butyric acid, tests for, 362
Buzzard, 289

Calomel in diarrhœa, 141, 145
—— in gastric catarrh, 246
Capacity of stomach, 3
————, methods of determining, 3
————, variations in, 4
Carbolic acid, administration of, 144, 278
Cardiac disease, dyspepsia of, 350
Care of the milk, 37
Cascara, use of, 89
Cases of gastric ulcer, 288
Cassel, 74
Castor-oil in diarrhœa, 145
—— mixture, 368
Casts of the gastric glands, 224
Cerebral thrombosis, 123
Chantemesse, 217
Charcot, 297
Chemistry of digestion in acute gastric catarrh, 240
———— in chronic gastric catarrh, 191, 257
———— in dyspepsia, 74, 271
———— in infancy, 9
Cholera, infantile, 91
————, symptoms of, 123
————, treatment of, 148
Chronic gastric catarrh, 248
————, diagnosis of, 258
————, digestion in, 257
————, etiology of, 257
————, histology of, 250
————, morbid anatomy of, 249
————, symptoms of, 254
————, treatment of, 258
————, varieties of, 250
Chronic gastro-intestinal catarrh, 150
————, appetite in, 181
————, atrophy of intestine in, 166
————, atrophy of stomach in, 164
————, blood in, 193
————, cerebral disease in, 172
————, complications of, 185
————, convulsions in, 184
————, diagnosis of, 200
————, diarrhœa in, 176
————, digestion in, 191
————, etiology of, 151
————, fatty liver in, 169
————, fever in, 181
————, general symptoms of, 178
————, histology of, 159
————, kidney disease in, 170
————, morbid anatomy of, 155
————, physical signs of, 190
————, prognosis in, 197
————, progress of, 195
————, sequelæ of, 187
————, thirst in, 182
————, treatment of, 202
————, ulcer of intestine in, 158
————, vomiting in, 174
Chvostek, 294
Clark, 319
Colgar, 290
Congenital stenosis of pylorus, 315
Constipation, treatment of, 88, 277
Contra-indications to nursing, 27
Convulsions in gastric catarrh, 229
—— in intestinal catarrh, 123, 184

Convulsions in dyspepsia, 72
——, treatment of, 88
Cough, reflex, 73
Cream mixtures, 41, 43, 367
Creasote, uses of, 144, 210
Cruveilhier, 283
Cullingworth, 307
Cyclical vomiting, 233
Czerny, 112

Daettwyler, 304
Decker, 216
Demme, 69
Development of stomach, 1
Diarrhœa, antiseptics in, 88
——, fat, 69
——, green, 70, 112
——, inflammatory, 91
——, lienteric, 176
——, simple, 59, 68
——, summer, 94
——, zymotic, 91
Diet in acute gastric catarrh, 242, 245
—— in acute intestinal catarrh, 135
—— in chronic gastric catarrh, 260
—— in chronic intestinal catarrh, 204
—— in dyspepsia, 82
—— in infancy and childhood, 32, 43, 55, 56, 57
—— in gastric ulcer, 317
—— in lactation, 30
—— in strumous dyspepsia, 331
—— in weak digestion, 272, 276
Differences between cow's milk and human milk, 36
Digestion, chemistry of, in acute gastric catarrh, 240
——, ——, in chronic gastric catarrh, 191, 257
——, ——, in dyspepsia, 74
——, ——, in kidney disease, 356
——, ——, in paroxysmal hyperacidity, 338
——, ——, in phthisis, 348
——, ——, in weak digestion, 271
——, normal process of, 11
Dilatation of stomach from chronic catarrh, 156
———— in gastric ulcer, 315
———— in stenosis of pylorus, 315
Dilution of milk, 38
Donné, 300
Dried milks, 47
Duclaux, 113
Duodenal indigestion, 69
Duodenitis, 108, 298
Dyspepsia in infancy, 59
——, appetite in, 71
Dyspepsia in infancy—*Cont.*
——, asthma in, 73
——, bronchitis in, 78
——, complications of, 77
——, diagnosis of, 79
——, diarrhœa in, 68
——, diet in, 82
——, etiology of, 59
——, frequency of, 59
——, symptoms of, 65
——, treatment of, 80
Dyspepsia in heart disease, 350
—— in nervous disease, 357
—— in phthisis, 343
—— in renal disease, 353
——, secondary forms of, 343
——, strumous, 319
——, ——, acidity in, 327
——, ——, anæmia in, 327
——, ——, appetite in, 325
——, ——, cascara in, 332
——, ——, climate for, 331
——, ——, cod-liver-oil in, 333
——, ——, constipation in, 326
——, ——, diarrhœa in, 327
——, ——, diet in, 331
——, ——, etiology of, 329
——, ——, flatulence of, 327
——, ——, frequency of, 320
——, ——, gastritis in, 320
——, ——, iron in, 333
——, ——, nausea in, 327
——, ——, pain in, 322
——, ——, purgatives in, 332
——, ——, symptoms of, 322
——, ——, thirst in, 326
——, ——, tongue in, 327
——, ——, treatment of, 33

Eczema in dyspepsia, 78
—— in intestinal catarrh, 126
Elsässer, 105
Emaciation in acute gastric catarrh, 119
—— in chronic gastric catarrh, 178
—— in dyspepsia, 72
—— in gastric ulcer, 314
Embarras gastrique, 226
Emetics in dyspepsia, 85
—— in gastric catarrh, 244
Enteric fever, ulcer of stomach in, 286
Epidemic gastric catarrh, 217, 239
—— intestinal catarrh, 94
Epithelium, detachment of, in intestine, 108
Epstein, 126
Erosions, hæmorrhagic, 281
Eross, 293
Escherich, 14, 16
Etiology of acute gastric catarrh, 213

Etiology of acute gastro-intestinal catarrh, 92
—— of chronic gastric catarrh, 248
—— of chronic gastro-intestinal catarrh, 151
—— of dyspepsia, 59, 262
—— of gastric ulcer, 295, 301
—— of hyperacidity, 340

Farinaceous foods in dyspepsia, 64
Fat diarrhœa, 69
Fatty degeneration of heart, 171
———— of kidney, 169
———— of liver, 171
Febrile gastric catarrh, 238
Feeding apparatus, 51
Feeding, mixed, 29
Fettmilch, 41
Finkelstein, 316
Fitful vomiting, 233
Fleishmann, 3
Flügge, 113
Follicular ulcers of intestine, 127, 158
———— of intestine, treatment of, 210
———— of stomach, 283
————, appearances of, 283
————, causes of, 283
————, pathology of, 283
————, results of, 286
Food, amount of, in infancy, 48
—— sterilization of, 49
Foods, artificial, 33
——, malted, 54
——, pancreatized, 54
——, predigested, 53
Fox, 220
Fränkel, 230
Frankland's artificial human milk, 40
Frequency of meals in infancy, 47
Frolowsky, 3

Gaertner's Fettmilch, 41
Gaffky, 217
Gases of stomach, 17
Gastric catarrh, acute, 213, 226
————, chronic, 248
————, subacute, 230
Gastric fever, 240
Gastric glands, casts of, 224
Gastric secretion in acute gastric catarrh, 240
———— in chronic gastric catarrh, 257
———— in dyspepsia, 74, 271
———— in infancy, 9
Gastric ulcer, 280
————, etiology of, 295
————, symptoms of, 308
————, treatment of, 316
————, varieties of, 281

Gastritis, acute interstitial, 221
——, acute parenchymatous, 222
——, chronic interstitial, 159, 251
——, chronic parenchymatous, 250
—— in kidney disease, 354
——, membranous, 220
——, toxic, 236
Gastro-duodenitis in burns, 297
————, etiology of, 298
Gastro-intestinal antisepsis, 140
Gastro-intestinal catarrh, acute, 91
————, chronic, 150
Gastroxie, 339
Gastroxynsis, 339
Gavage, 134, 204
Gelatinous softening of stomach, 103
Goat's milk, 34
Goodhart, 289
Green diarrhœa, etiology of, 70
————, treatment of, 142
Grimshaw, 217
Gundobin, 193
Gunz, 292

Hæmatemesis, causes of, 310
—— in gastric ulcer, 311
Hæmoglobin in marasmus, 194
Hæmorrhage from bowel, 175
————, treatment of, 318
Hæmorrhagic erosions, 281
————, appearances of, 282
————, causes of, 281
————, results of, 282
Hayem, 70, 111, 193
Heart disease, dyspepsia of, 350
Heart, fatty degeneration of, 171
Henoch, 229, 230
Henschel, 3
Hirschsprung, 126, 315
Histology of infant's stomach, 4
Holt, Emmett, 3, 95, 100, 108, 126
Hydrochloric acid, secretion of, 9, 10
———— in acute gastric catarrh, 240
———— in chronic gastric catarrh, 191, 257
———— in dyspepsia, 74, 271
————, estimation of, 363
————, tests for, 362
————, uses of, 90, 149, 247, 261
Hyperacidity, causes of, 334
——, paroxysmal, 334

Idiosyncrasies of digestion, 213
Indigestion in infants, 59
Infant feeding, 32
——, weight of, 26
Infantile atrophy, 150
Infective gastro-intestinal catarrh, 94
Inflammatory diarrhœa, 91

Influence of mother's health on her milk, 23
Interstitial gastritis, 221
Intestinal antisepsis, 140
—— hæmorrhage, 175, 311
Intestine in acute catarrh, 102, 109
—— in chronic catarrh, 157
——, cirrhosis of, 167, 169
——, follicular ulceration of, 102
——, gases of, 16
——, softening of, 104
——, irrigation of, 134, 211
Intussusception in acute intestinal catarrh, 127
Iodoform, uses of, 143
Ipecacuanha, uses of, 85, 147, 210
Irrigation of intestine, 211

Jackson, 308
Jeffries, 70, 112

Kaulich, 307
Kéfir in diarrhœa, 136, 208
Kidney disease, dyspepsia of, 353
Kidney, fatty degeneration of, 169
——, secondary inflammation of, 126
——, thrombosis of, 170
——, uratic concretions in, 171
Kjellberg, 126
Klebs, 297
Korowin, 14
Koumiss in diarrhœa, 136, 208

Lactic acid, detection of, 362
————, estimation of, 364
————, formation of, 362
————, use of, in diarrhœa, 142
Landau, 312
Landerer, 315
Lavage in chronic gastritis, 133, 203
Lebert, 280
Legendre, 171
Leith, 294
Leo, 12, 74
Lepine, 339
Lesage, 70, 100, 111
Liver, fatty degeneration of, 171
Loewy, 65
Loschner, 220
Lubert, 113
Lüttke, 12

Maier, 315
Malinowski, 297
Malted foods, 54
Mammary secretion, 18
————, variations of, 20
Marasmus, 150
Measles, stomach in, 225
Meinert, 97
Melæna, causes of, 311
—— in gastric ulcer, 311
—— neonatorum, 312
——, pathology of, 312
——, symptoms of, 313
——, treatment of, 318
Mellin's food, 55
Membranous gastritis, 217
Mercuric chloride in diarrhœa, 144, 145
Milk, 18
——, analysis of, 19, 46, 33
——, artificial human, 40
——, ass's, 34
——, bacteria of, 113
——, care of, 37
——, condensed, 44
——, cow's, 34, 36
——, dilution of, 38
——, dried, 47
——, human, 18
——, intolerance of, 52
——, modification of, 38
——, predigestion of, 53
——, secretion of, 18
——, sterilization of, 49
——, variations in composition of, 20
Miller, 12, 97, 126
Mixed feeding, 29
Mixtures, cream, 41, 367
Mortality from summer diarrhœa, 97
Mozer, 112

Naphthalin, uses of, 88, 143
Nausea in dyspepsia, 66
—— in gastric catarrh, 232
—— in weak digestion, 266
Nervous disease, dyspepsia in, 357
Nervous symptoms in acute gastric catarrh, 229
———— in chronic gastro-intestinal catarrh, 184
———— in dyspepsia, 72
———— in weak digestion, 269
Neuretter, 220
Non-healing of gastric ulcers, 301
Nursing, contra-indications to, 27
——, rules of, 124

Opium in dyspepsia, 88
—— in gastric ulcer, 317
—— in intestinal catarrh, 146
Otto, 193

Pain in chronic intestinal catarrh, 177
—— in dyspepsia, 65
—— in gastric ulcer, 309
—— in strumous dyspepsia, 322
Pancreas, secretion of, 14

Pancreatized foods, 54
Parenchymatous gastritis, acute, 222
————, chronic, 250
Paroxysmal hyperacidity, 334
————, symptoms of, 335
————, treatment of, 341
Parrot, 157, 159, 169, 171, 172, 173, 220
Pasteurization of milk, 49
Paulicki, 306
Peden, 315
Pedley, Denison, 264
Pepsin, secretion of, 10
—— in chronic gastric catarrh, 183, 257
—— in chronic dyspepsia, 90, 279
Pericarditis in chronic gastro-intestinal catarrh, 186
Periodic vomiting, 233
Pfannenstill, 13
Pfeiffer, 70
Pfungen, 11
Phthisis, dyspepsia of, 343
——, ——, treatment of, 348
Physiology of stomach in infancy, 9
Pitt, 307, 315
Pleurisy in intestinal catarrh, 126
Post-mortem digestion of stomach and bowel, 103
Preserved milks, 44
————, objections to, 45
————, employment of, 45
Prognosis in acute gastro-intestinal catarrh, 130
—— in chronic gastro-intestinal catarrh, 197
Prolapse of rectum in diarrhœa, 147
Ptyalin, secretion of, 13
Pulmonary disease, dyspepsia in, 343
Pulmonary tuberculosis in chronic intestinal catarrh, 172
Purgatives in acute intestinal catarrh, 140
—— in constipation, 86
—— in weak digestion, 277
Puteren, 12, 14, 74
Pylorus, position of, in infancy, 2
——, congenital stenosis of, 315
——, stenosis of, from ulcer, 315

Raw meat pulp, preparation of, 366
————, use of, 207
Rayer, 220
Recipes, 365
Reflex symptoms in dyspepsia, 73, 268
Refusal of breast, 26
Rehn, 289
Reimer, 292
Renal disease, dyspepsia of, 353
—— in intestinal catarrh, 126
Resorcine in diarrhœa, 88, 144, 146
—— in dyspepsia, 278
Rickets a cause of wasting, 201
—— a result of chronic intestinal catarrh, 187
Rilliet, 153, 305, 313
Rokitansky, 270, 280
Rosenthal, 339
Rossbach, 339
Rotch, 3, 42, 48
Rules of nursing, 24

Saburral condition, 213
Salicylates, uses of the, 142,
Saline injections in collapse, 133
Saliva in infancy, 13
Salol, use of, 142
Savelieff, 239
Savory and Moore's food, 55
Scarlatinal gastritis, 223
Scheffer, 307
Scheme of diet in infancy, 43, 55, 56, 57
———— in gastric catarrh, 245, 260
———— in weak digestion, 276
Sclerema in intestinal catarrh, 128
Secondary gastric catarrh, 217
—— varieties of dyspepsia, 343
Siebert, 97
Siegmund, 229
Silbermann, 304, 313
Simple diarrhœa, 68
Simple gastric ulcer, 287
Simple intestinal catarrh, 92
Size of meals in infancy, 47
Skin affections in dyspepsia, 78
———— in intestinal catarrh, 128 180
———— in weak digestion, 269
Snow, 339
Solitary glands of stomach, 6
————, disease of, 283
Spurious hæmatemesis, 310
Status gastricus, 213
Steiner, 220, 305
Stenosis of pylorus, congenital, 315
————, from ulcer, 315
Sterilization of milk, 49
Sterilizer, the, 51
Stomach, anatomy of, 1
——, atony of, 75, 271
——, atrophy of, 164
——, bacteria of, 14
——, capacity of, 3, 4
——, catarrh of, 101, 160, 213, 248
——, chemical examination of, 361
——, dilatation of, 156, 315
——, development of, 1
——, gases of, 17
——, growth of, 3

Stomach, histology of, 4
——, malignant disease of, 307
——, motor activity of, 12
——, physiology of, 9
——, position of, 2
——, softening of, 103
——, tuberculosis of, 305
——, ulceration of, 280
Stomatitis in dyspepsia, 78
—— in gastric catarrh, 128
Stools, examination of, 117
Strippings, 52
Strophulus in dyspepsia, 78
Strumous dyspepsia, 319
Subacute gastric catarrh, 226
Suckling, rules of, 24
Summer diarrhœa, 94, 97
————, bacteriology of, 110

Teats, choice of, 52
Temperature of air in epidemic diarrhœa, 97
Tetany of intestinal origin, 124
Thompson, 316
Thomson, 220, 240
Thrombosis of cerebral veins, 123, 172
—— of renal veins, 170
Todd, 319
Tomkins, 112
Tongue, ulceration of, in gastric catarrh, 232
Tonics in dyspepsia, 90
—— in gastric catarrh, 247
Toxic gastritis, 216
Transfusion in collapse, 148
Trypsin, secretion of, 14
Tubercular ulcer of stomach, 305
Tuberculosis, emaciation in, 201
—— in chronic intestinal catarrh, 187
Turpentine in infantile convulsions, 88
—— in melæna neonatorum, 318
Tyrotoxicon in milk, 113

Ueffelmann, 65
Ulceration, follicular, of intestine, 102, 158
Ulceration of stomach, 280
————, acute perforating, 288
————, cases of, 288
————, chronic, 292
————, complications of, 314
————, etiology of, 295
————, follicular, 283
————, frequency of, in childhood, 280
————, hæmatemesis in, 310
————, hæmorrhagic, 281
———— in burns, 297
Ulceration of stomach—*Continued.*
————, malignant, 307
————, melæna in, 311
————, non-healing of, 301
————, symptoms of, 308
————, treatment of, 316
————, varieties of, 281
Uratic concretions in kidney, 171
Uræmia, dyspepsia of, 356
—— in acute intestinal catarrh, 125

Vacher, 98
Vaughan, 113
Vidal, 220
Vomiting, causes of, 67
—— in acute gastric catarrh, 114, 227
—— in chronic gastric catarrh, 176, 254
—— in dyspepsia, 68
—— in gastric ulcer, 309
—— in heart disease, 351
—— in kidney disease, 356
—— in nervous disease, 358
—— in phthisis, 344
—— in uræmia, 356
——, periodic, 232
Vomitus acidus, 339

Wasting in gastro-intestinal catarrh, 119, 178
—— in ulceration of the stomach, 314
Weak digestion, 262
————, asthma in, 270
————, cardiac symptoms in, 269
————, constipation in, 267
————, cutaneous phenomena in, 269
————, diet in, 272, 276
————, digestion in, 271
————, etiology of, 262
————, nervous symptoms in, 269
————, primary, 262
————, secondary, 265
————, symptoms of, 265
————, treatment of, 271
Weaning, 29
——, diet after, 30
Weight of infant at different ages, 26
Welch, 280
Wertheimber, 300
West, 171
Wet-nurse, 32
Whortleberries in diarrhœa, 207
Widerhofer, 305
Williamson, 308, 315
Willigk, 215

Zweifel, 14
Zymotic diarrhœa, 91

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

P446 Fenwick, W.S. 90355
F34 The disorders of
1897 digestion in infancy
and childhood.

NAME	DATE DUE